A COMPARATIVE STUDY ON ANALGESIC EFFICACY OF BILATERAL SUPERFICIAL CERVICAL PLEXUS BLOCK WITH 0.25% BUPIVACAINE AND 0.25% BUPIVACAINE WITH CLONIDINE FOR THYROIDECTOMY SURGERIES

Dissertation Submitted in partial fulfillment of

M.D. DEGREE EXAMINATION

M.D. ANAESTHESIOLOGY- BRANCH X

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THE TAMILNADU DR. M. G. R. MEDICAL UNIVERSITY

CHENNAI, TAMILNADU

APRIL 2011

CERTIFICATE

This is to certify that this dissertation titled **"A COMPARATIVE STUDY ON ANALGESIC EFFICACY OF BILATERAL SUPERFICIAL CERVICAL PLEXUS BLOCK WITH 0.25% BUPIVACAINE AND 0.25% BUPIVACAINE WITH CLONIDINE FOR THYROIDECTOMY SURGERIES"** has been prepared by **Dr.Nimisha Checha Jacob** under my supervision in the Department of Anaesthesiology, Chengalpattu Medical College and Hospital, Chengalpattu during the academic period 2008-2011 and is being submitted to the Tamilnadu Dr. M. G. R. Medical University, Chennai in partial fulfillment of the University regulations for the award of the Degree of Doctor of Medicine (Branch X – M.D. Anaesthesiology) and her dissertation is a bonafide work.

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DECLARATION

I, **Dr. Nimisha Checha Jacob,** solemnly declare that the dissertation **"A COMPARATIVE STUDY ON ANALGESIC EFFICACY OF BILATERAL SUPERFICIAL CERVICAL PLEXUS BLOCK WITH 0.25% BUPIVACAINE AND 0.25% BUPIVACAINE WITH CLONIDINE FOR THYROIDECTOMY SURGERIES"** is a bonafide work done by me in the Department of Anaesthesiology, Chengalpattu Medical College & Hospital, Chengalpattu, after getting approval from the Ethical Committee, under the able guidance of PROF. DR. N.KRISHNAN, M.D.,D.A, Professor and Head of the Department of Anaesthesiology, Chengalpattu Medical College, Chengalpattu.

Place: Chengalpattu,

Date: **Dr. Nimisha Checha Jacob**

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CONTENTS

AIM OF THE STUDY

To evaluate the quality and duration of postoperative analgesia with bilateral superficial cervical plexus block for patients undergoing thyroidectomy surgeries under general anaesthesia using 0.25% **B**upivacaine (Group **B**) and 0.25% Bupivacaine with **C**lonidine 5 μg/ml (Group **C**)

INTRODUCTION

Pain is a protective mechanism designed to alert the body to potentially injurious stimuli. The International Association for the Study of Pain (IASP) defines pain as an unpleasant sensory and emotional experience associated with actual or potential tissue damage. Alleviation of pain has been the focus of continuing human effort over centuries.

Thyroid operations can cause mild to moderate incisional pain. In addition, discomfort in swallowing, burning sensation in the throat, nausea, and vomiting can be caused by the operation or by general anaesthesia. These affect a majority of the patients, especially within the first day after operation. Surgeons and anaesthesiologists have attempted to prevent or treat these problems with various modalities, such as opioids and nonsteroidal antiinflammatory drugs, or with additional locoregional anaesthesia techniques. Locoregional anaesthesia, such as local anaesthetic wound infiltration[21], bilateral superficial cervical plexus block (BSCPB)[5], and bilateral combined superficial and deep cervical plexus block[3], can potentially reduce postoperative pain in patients who undergo thyroidectomy.

Bilateral superficial cervical plexus block (BSCPB) is a popular regional anaesthesia technique for its feasibility and efficacy. Clonidine,

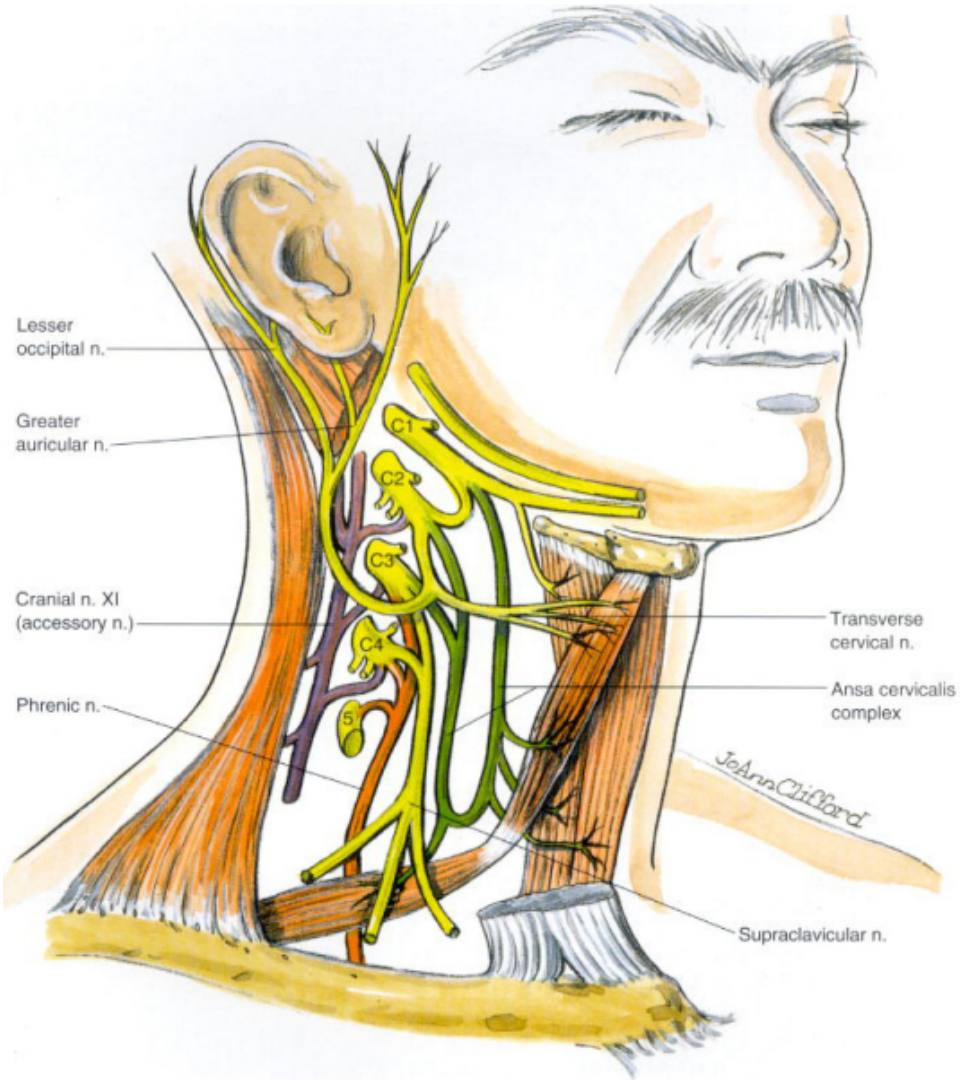
an alpha-2 adrenergic agonist, has been used as an adjuvant to local anaesthetics in various regional techniques[9,12,13] to extend the duration of block. This study was conducted to see the efficacy of Clonidine as an adjunct to Bupivacaine in post operative pain relief following bilateral superficial cervical plexus block for thyroid surgeries. All patients undergoing thyroidectomy surgeries under general anaesthesia, received bilateral superficial cervical plexus block using 6ml of 0.25% Bupivacaine (Group B) or 6ml of 0.25% Bupivacaine with Clonidine 5 μg/ml (Group C) on each side.

CERVICAL PLEXUS

The anterior rami of the upper four cervical nerves unite by a series of loops to form the cervical plexus, whose function is the supply of the skin and muscles of the neck and the innervation of the diaphragm.

The branches of the cervical plexus can be divided into four groups.

1. Communicating branches, which pass to the hypoglossal nerve, and which also pass to the vagus and to the cervical sympathetic chain.
2. Superficial branches, which supply cutaneous fibres to the neck.
3. Deep branches, to the neck muscles.
4. The phrenic nerve, which is the motor nerve of the diaphragm


Lesser occipital n.
Greater auricular n.
Cranial n. XI (accessory n.)
Phrenic n.
C1
C2
C3
C4
5
Transverse cervical n.
Ansa cervicalis complex
Supraclavicular n.
JoAnn Clifford

THE SUPERFICIAL CERVICAL PLEXUS

The superficial cervical plexus comprises superficial branches that can be tabulated into ascending, transverse and descending groups.

1 Ascending - Lesser occipital nerve (C2); great auricular nerve (C2, 3)

2 Transverse - Anterior cutaneous nerve of neck (C2, 3)

3 Descending - Supraclavicular nerves (C3, 4)

The lesser occipital nerve (C2) hooks around the spinal accessory nerve (XI), and then ascends along the posterior border of the sternocleidomastoid. It pierces the deep fascia in the upper part of the posterior triangle, and then splits up into three branches.

1. Auricular - to the upper third of the medial aspect of the external ear
2. Mastoid - to the skin over the mastoid process
3. Occipital - to the occipital area immediately above and behind the mastoid

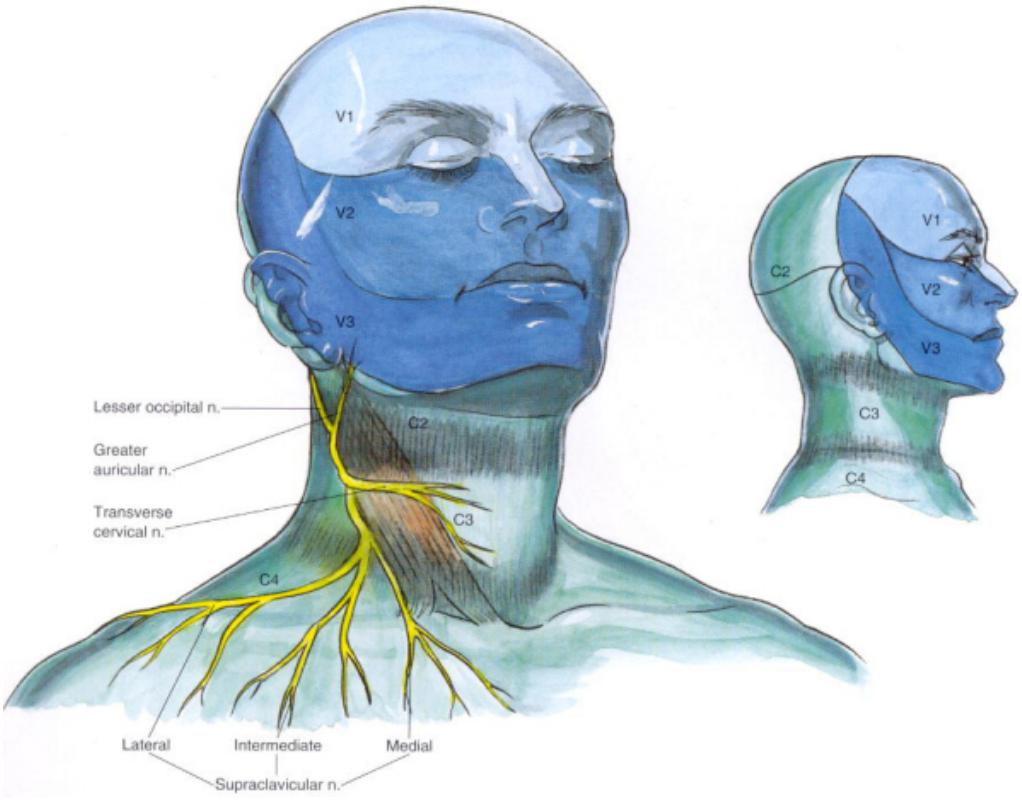
The great auricular nerve (C2, 3) is the largest cutaneous branch of the cervical plexus. It hooks around the mid-point of the posterior border

of sternocleidomastoid, and then passes across it in the direction of the angle of the mandible. On this muscle it breaks up into three terminal branches.

1. Auricular - supplying the lower two-thirds of the medial aspect of the external ear and the lateral surface of the lobule.
2. Mastoid - to the skin over the mastoid process
3. Facial -to the skin over the masseter and the parotid gland

The anterior cutaneous nerve of the neck (C2, 3) emerges close below the great auricular nerve at the posterior border of sternocleidomastoid, then passes horizontally forward on the muscle, deep (sometimes superficial) to the external jugular vein. At the anterior border of sternocleidomastoid, the nerve pierces the deep fascia and splits into branches to supply the skin of the whole front of the neck.

The supraclavicular nerves (C3, 4) arise as a common stem that emerges from behind sternocleidomastoid immediately below the other cutaneous nerves of the plexus. This stem soon splits into three branches - medial, intermediate and lateral, which pierce the deep fascia above the clavicle, cross this bone and supply the skin over the upper sternum, the upper chest wall.


V1
V2
V3
Lesser occipital n.
Greater auricular n.
Transverse cervical n.
C2
C3
C4
Lateral
Intermediate
Medial
Supraclavicular n.
V1
C2
V2
V3
C3
C4

THE DEEP CERVICAL PLEXUS

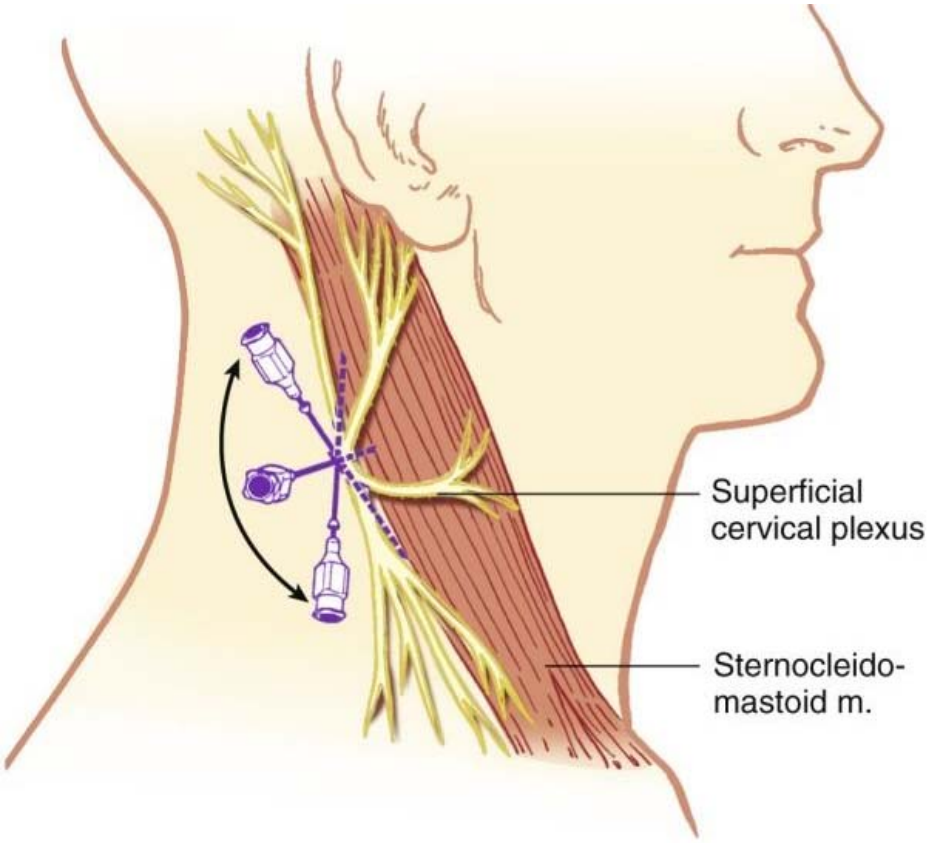
This supplies the anterior vertebral muscles - the recti capitis, longus capitis and longus cervicis, as well as giving contributions to scalenus medius. In addition, branches pass to levator scapulae (C3, 4) and to two muscles whose principal innervation is from the spinal accessory nerve: sternocleidomastoid (C2, 3) and trapezius (C3, 4).

CLINICAL APPLICATIONS

Blockade of the cervical plexus is easy to perform and provides anaesthesia for surgical procedures in the distribution of C2 to C4, including lymph node dissection, plastic repair, and carotid endarterectomy. Bilateral blocks can be used for tracheostomy and thyroidectomy. Cervical plexus block can be divided into superficial and deep techniques.

SUPERFICIAL CERVICAL PLEXUS BLOCK

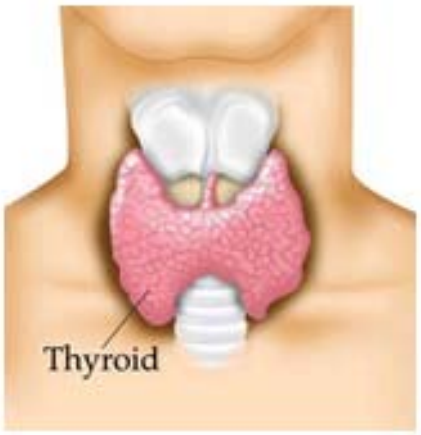
The patient is placed in the supine position, with the head and neck turned opposite the side to be blocked. The anaesthesiologist should stand at the patient's side approximately shoulder high. At the midpoint on the posterior border of the sternocleidomastoid muscle, the superficial cervical plexus is packaged so infiltration at this point produces a block. To perform the block, a 24-gauge, 2.5cm needle is inserted subcutaneously posterior to the sternocleidomastoid muscle, and 2 mL of local anaesthetic is injected. The needle is redirected both superiorly and inferiorly along the posterior border of the sternocleidomastoid, and 2 mL of solution is injected along each of these sites. A total of 6 mL of local anaesthetic is thus injected. In this fashion, a field block of the superficial plexus is created. If patients are properly positioned for this block, the superficial block rarely results in problems. Complications from improper performance of the block include a deeper placement of needle leading to deep cervical plexus block, which includes temporary Horner's syndrome, phrenic nerve paralysis, and hoarseness from recurrent laryngeal nerve paralysis. A potential for systemic toxicity is present because of injection of the local anaesthesia solution into the vertebral artery or the external or internal jugular veins[24]


Superficial
cervical plexus
Sternocleido-
mastoid m.

THE THYROID GLAND

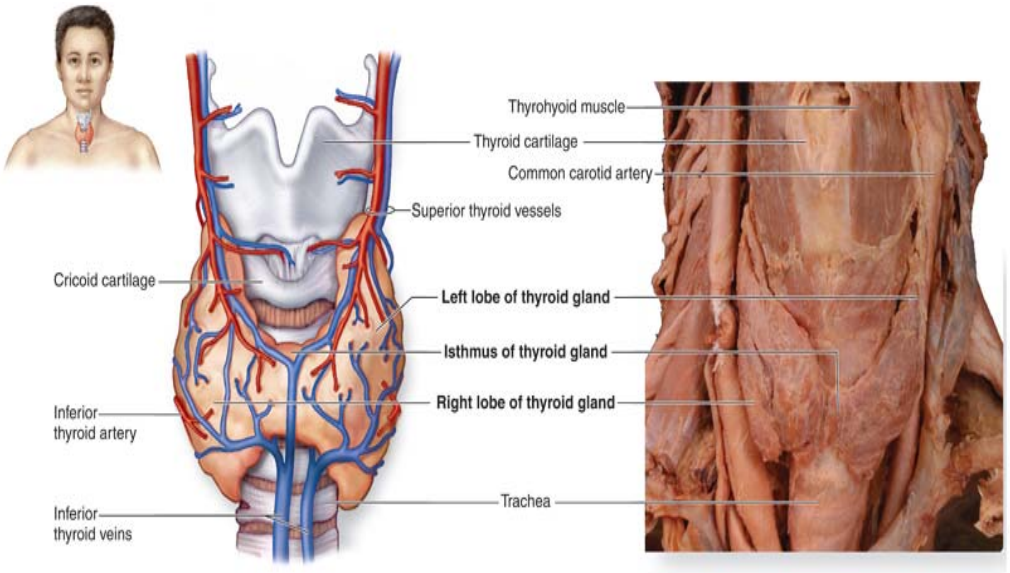
Anatomy

Although the term thyroid is derived from the Greek word meaning shield, the gland is most commonly described as ‘butterfly’ shaped. The thyroid gland lies in the neck related to the anterior and lateral parts of the larynx and trachea. Anteriorly, its surface is convex; posteriorly, it is concave. It is composed of two lobes joined by an isthmus. The isthmus lies across the trachea anteriorly just below the level of the cricoid cartilage. The lateral lobes extend along either side of the larynx as roughly conical projections reaching the level of the middle of the thyroid cartilage. Their upper extremities are known as the upper poles of the gland. Similarly, the lower extremities of the lateral lobes are known as the lower poles. The gland is brownish- red due to a rich blood supply.



Innervation of the Thyroid

Principal innervation of the thyroid gland derives from the autonomic nervous system. Parasympathetic fibers come from the vagus nerves, and sympathetic fibers are distributed from the superior, middle, and inferior ganglia of the sympathetic trunk. These small nerves enter the gland along with the blood vessels. Autonomic nervous regulation of the glandular secretion is not clearly understood, but most of the effect is postulated to be on blood vessels, hence the perfusion rates of the glands.



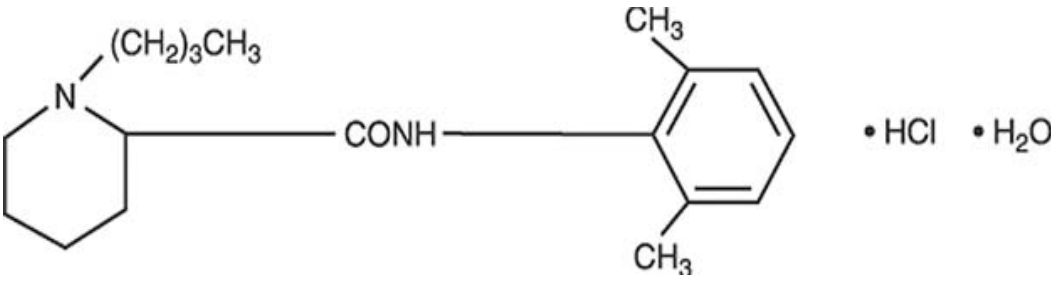
PHARMACOLOGY OF BUPIVACAINE

Bupivacaine is a local anaesthetic drug belonging to the amino amide group, synthesised by A.F.Ekenstam in 1957 and brought into clinical use in 1963.

It is produced for clinical use in a racemic mixture containing equal proportions of ‘S’ and ‘R’ enantiomers. It is supplied for clinical use as hydrochloride salt.

CHEMICAL STRUCTURE

Bupivacaine hydrochloride is (±) -1-Butyl-2′,6′-pipecoloxylidide monohydrochloride, monohydrate.



Physicochemical and pharmacokinetic Profile[26]

Molecular weight	-	288
pKa	-	8.1
Plasma protein binding	-	95%
Liposolubility	-	30

Partition coefficient (octanol/buffer) - 3460

Volume of distribution at steady state – 73 L

T 1/2 (min) - 210

Clearance (L min-1) - 0.58

Mechanism of action

Bupivacaine exerts its effect by inhibition of sodium channels. Bupivacaine binds to the intracellular portion of sodium channels and blocks sodium influx into nerve cells, which prevents depolarization. It acts to block conduction in nerves by decreasing or preventing the large transient increases in permeability of cell membrane to sodium ions that follows depolarisation of the membrane. Bupivacaine also reduces the permeability of the resting nerve membrane to potassium as well as sodium ions.

Pharmacokinetics

Bupivacaine is rapidly absorbed from the site of injection. The rate of rise of plasma bupivacaine concentration and the peak plasma concentration depends on the route of administration. There is also interindividual variation and peak systemic concentration may occur 5 and 30 mins after administration. The addition of vasoconstrictor delays absorption and results in lower plasma concentrations of bupivacaine.

Pharmacokinetic studies on the plasma profiles of bupivacaine after direct intravenous injection suggest a three-compartment open model. The first compartment is represented by the rapid intravascular distribution of the drug. The second compartment represents the equilibration of the drug throughout the highly perfused organs such as the brain, myocardium, lungs, kidneys and liver. The third compartment represents an equilibration of the drug with poorly perfused tissues, such as muscle and fat. The elimination of drug from tissue distribution depends largely upon the ability of binding sites in the circulation to carry it to the liver where it is metabolized.

Metabolism

Bupivacaine is metabolized primarily in the liver via conjugation with glucuronic acid. Pipecoloxylidine is the major metabolite of bupivacaine. Half life is 3.5 hours in adults and 8.1 hours in neonates. The kidney is the main excretory organ for bupivacaine and its metabolites. Urinary excretion is affected by renal perfusion and factors affecting urinary pH. Only 5 - 10% of bupivacaine is excreted unchanged in the urine.

Clinical applications

- Infiltration anaesthesia
- Peripheral nerve block
- Central neuraxial block (intrathecal, epidural, caudal)

Contraindications

- Known hypersensitivity to bupivacaine or to any local anaesthetic agent of the amide type
- Intravenous regional anaesthesia

Preparations available

- 0.25%, 0.5% in 10 and 20ml vials containing methyl paraben as preservative
- 5mg/ml (0.5%) bupivacaine with 80mg dextrose in 4ml ampoules for intrathecal injection

Adverse reactions

Adverse reactions are associated with excess plasma levels of the drug which may be due to over dosage, unintentional intravascular injection or slow metabolic degradation.

Bupivacaine is more cardiotoxic. Clinically, this is manifested by severe ventricular arrhythmias and myocardial depression after inadvertent intravascular administration of large doses of bupivacaine. The enhanced cardiotoxicity of bupivacaine probably is due to multiple factors. Bupivacaine blocks cardiac sodium channels rapidly during systole. However, bupivacaine dissociates much more slowly during diastole, so a significant fraction of sodium channels remain blocked at the end of diastole with bupivacaine. Thus, the block by bupivacaine is cumulative and substantially greater than would be predicted by its local anesthetic potency. Bupivacaine-induced cardiotoxicity can be very difficult to treat, and its severity is enhanced in the presence of acidosis, hypercarbia, and hypoxemia.

The central nervous system effects are characterized by excitation or depression. The first manifestation may be nervousness, dizziness, blurred vision, or tremors, followed by drowsiness, convulsions, unconsciousness, and cardiac arrest.

The ratio of the dosage required for irreversible cardiovascular collapse (CC) and the dosage that will produce CNS toxicity (convulsions) (i.e., the CC/CNS ratio) is lower for bupivacaine.

Allergic reactions, which may be due to hypersensitivity, idiosyncrasy, or diminished tolerance, are characterized by cutaneous lesions (e.g. urticaria), edema, and other manifestations of allergy.

The clinical implications for cardiac resuscitation after intravascular injection or overdose of local anesthetic are the following:

- No medications are uniformly effective in facilitating resuscitation from bupivacaine-induced cardiac arrest or severe ventricular tachycardia. Basic principles of cardiopulmonary resuscitation should be emphasized first, including attention to securing the airway, providing oxygenation and ventilation, and performing chest compressions if needed.

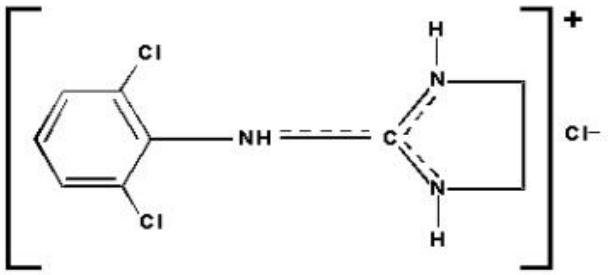
- If a patient experiences profound cardiovascular depression or circulatory arrest after the administration of bupivacaine, then along with initiation of basic life support and the ACLS protocol a rapid bolus of Intralipid 20%, 1.5 mL/kg, be administered without delay, followed if necessary by an infusion of 0.25 mL/kg/min for the next 10 minutes.

PHARMACOLOGY OF CLONIDINE

Clonidine is a centrally acting selective alpha-2 adrenergic agonist. It has a ratio of 220:1 alpha-2 selectivity (alpha-2: alpha-1). It is an imidazole derivative and was formulated in 1960, initially as a vasoconstrictor and nasal decongestant.

It acts primarily by reducing the sympathetic output from the central nervous system.

The chemical name is 2-(2,6-dichlorophenylamino)-2-imidazoline hydrochloride. The following is the structural formula:



$C_9H_9Cl_2N_3 \cdot HCl$ Mol. Wt. 266.56

Mechanism of action

Clonidine binds with alpha-2 adrenergic receptors which are of three subtypes – Alpha 2A, 2B, and 2C. Alpha 2A receptors mediate sedation, analgesia and sympatholysis. Alpha 2B receptors mediate

vasoconstriction and anti-shivering effects whereas Alpha 2C mediate the startle response.

Highest densities of alpha - 2 receptors are present in the pontine locus ceruleus, an important source of sympathetic nervous system innervation of the forebrain and a vital modulator of vigilance. Clonidine stimulates alpha-2 adrenergic inhibitory neurons in the medullary vasomotor center. There is a decrease in the sympathetic nervous system outflow from central nervous system to peripheral tissues, manifested as peripheral vasodilatation and decrease in systemic blood pressure, heart rate and cardiac output. The ability to modify the functions of potassium channels in the CNS decreases anaesthetic requirements. Alpha-2 receptors on peripheral sympathetic nervous system nerve endings inhibit release of norepinephrine. Neuraxial placement of clonidine inhibits spinal substance P release and nociceptive neuron firing.

The mechanism whereby clonidine prolongs the duration of local anaesthetic blockade when injected into a nerve sheath remains speculative. The action of clonidine is more likely via a synergistic mechanism of action in combination with the local anaesthetic resulting in the prolonged effect[16]. Clonidine possibly enhances or amplifies the sodium channel blockade action of local anaesthetics by opening up the

potassium channels resulting in membrane hyperpolarization, a state in which the cell is unresponsive to excitatory input. Clonidine may cause local vasoconstriction.

Clonidine produces sedation by decreasing sympathetic nervous system activity and the level of arousal. The result is a calm patient who can be easily aroused to full consciousness. Drugs that activate GABA receptors produce a clouding of consciousness and can cause paradoxical agitation as well as tolerance and dependence.

EFFECTS ON ORGAN SYSTEMS

Cardiovascular system

The decrease in systolic blood pressure produced by clonidine is more prominent than the decrease in diastolic blood pressure. Homeostatic cardiovascular reflexes are maintained, thus avoiding problems of orthostatic hypotension or hypotension during exercise. Renal blood flow and glomerular filtration rate are maintained in the presence of clonidine therapy.

Respiratory system

Clonidine has minimal depressant effects on ventilation and does not potentiate ventilatory depressant effects of opioids.

Central nervous system

Sedation is one of the most consistent effects mediated by central alpha-2 receptors. Another characteristic effect is anxiolysis. It has potent analgesic action that cannot be reversed by naloxone, an opioid antagonist, indicating that clonidine and opioid mediate analgesia through independent receptor mechanism.

There is a 50% decrease in MAC of inhalational anaesthetics and decreased anaesthetic requirements of opioids. It also prevents shivering. Clonidine is also effective in suppressing signs and symptoms of withdrawal from opioids, benzodiazepines and ethanol.

Pharmacokinetics

Clonidine is rapidly absorbed after oral administration and reaches peak plasma concentrations within 60 – 90 minutes. Elimination half life of clonidine is between 9 and 12 hours, with approximately 50% metabolized in liver and the rest excreted unchanged in urine.

Adverse effects

The most common side effects produced by clonidine are sedation and xerostomia. Although dose-dependent adverse effects such as hypotension and sedation and idiosyncratic adverse effects such as

bradycardia do occur, clonidine does not induce profound respiratory depression and only mildly potentiates opioid-induced respiratory depression. The other side effects are constipation, dizziness, fluid retention, sleep disturbances, impotence, parotid swelling and depression.

Withdrawal syndrome which is characterised by a rapid rise in blood pressure, with marked blood pressure lability, symptoms such as headache, flushing, sweating, insomnia, agitations, emotional lability, tremor, nausea and vomiting presents 18 – 72 hours after the last dose of clonidine. The syndrome can be prevented by gradual withdrawal of clonidine treatment or by inhibiting peripheral sympathetic nervous activity, with alpha and beta adrenoreceptor antagonists.

Transient hypertension and bradycardia may occur after i.v. injection, caused by direct stimulation of peripheral vascular alpha-2 receptors; an alpha–1 agonist effect may also contribute.

Disturbance of cardiac impulse generation and conduction in the presence of pre existing SA and AV node disease can lead to symptomatic bradycardia and impairment of atrio ventricular nodal conduction.

USES

Premedication

Due to its sedative effect, clonidine can be used as a premedicant. It also decreases the requirement of anaesthetics. Alpha-2 adrenergic agonists reduce the dose of intravenous hypnotic needed for anaesthesia induction and orotracheal intubation and also reduce the MAC of co-administered volatile anaesthetic agents by 50%. Clonidine has been recommended as premedicant in a dose of 1-3 μg/kg.

Clonidine as a premedicant is particularly useful in:

- ✓ Hypertensive patients have swings in blood pressure during surgery and premedication with clonidine is useful in reducing this hyper-reactivity.
- ✓ Chronic pain and palliative care patients receiving large doses of opioids may require greater perioperative opioids. This can be markedly reduced by premedication with clonidine.
- ✓ Drug addicts and alcoholics with problems such as withdrawal symptoms. The increased sympathetic activity in cocaine users is well controlled by clonidine.

The hemodynamic effects of clonidine are both peripheral and central. Stimulation of sub endothelial receptors causes vasoconstriction. Clonidine reduces the tonic activity of the baroreflex, decreasing arterial pressure and causing bradycardia. In addition, clonidine depresses presynaptic sympathetic neurons in the lateral horn of the thoracic spinal cord. Consequently, intrathecal administration causes more marked hypotension than parenteral administration.

Clonidine decreases plasma catecholamine levels. Giving clonidine before anaesthesia decreases cardiac output, vascular resistance, cardiac preload, afterload and contractility. During anaesthesia, clonidine increases cardiac output by improving cardiac loading conditions. In addition alpha-2 adrenergic agonists prevent hypertension and tachycardia on intubation and during surgical stimulation. During recovery from anaesthesia, these agents prevent tachycardia and hypertension, decrease the incidence of shivering and reduce oxygen consumption.

Postoperative Analgesia and Regional anaesthesia

Alpha-2 adrenergic agonists inhibit transmission of nociceptive stimuli in the dorsal horn of the spinal cord. Moreover they increase the analgesic effect of opioids. They augment local anaesthetic blockade and

prolong their duration of action. Clonidine has been used by epidural, spinal, perineural, intraarticular and parenteral routes to supplement postoperative analgesia produced by local anaesthetics.

Central nerve blocks

Clonidine produces dose dependent analgesia when administered into the epidural or subarachnoid space. Activation of post synaptic receptors in substantia gelatinosa of the spinal cord is the presumed mechanism for analgesic effect in central neuraxial blockade.

Intrathecal clonidine produces analgesia of shorter duration but without the associated risk of respiratory depression or urinary retention as seen with opioid. In association with local anaesthetics the maximum dose of intrathecal clonidine is 1µg/kg. Giving clonidine with a local anaesthetic improves the quality and duration of the block, minimizes the pain of the tourniquet and prevents shivering. Caudal clonidine combined with local anaesthetics in children is very useful and increases the duration of anaesthesia and analgesia by a factor of 2 or 3 without hemodynamic side effects. The dose is between 2 and 3 µg/kg.

Peripheral nerve blocks

Clonidine is commonly used as an adjuvant to local anaesthetics in peripheral nerve blocks where it prolongs the duration of anaesthesia and analgesia. Adding clonidine gives very good quality of analgesia after surgery with duration of over 24 hours for some lower limb blocks[28]. However there is a risk of prolonged motor block, particularly in the elderly patient which could prevent mobilisation. The quality of intravenous regional anaesthesia produced by lidocaine is improved by adding 150µg of clonidine. It particularly improves the tolerance of the tourniquet.

Contraindications

Disorders of cardiac impulse generation and conduction like sino atrial disease and atrioventricular node disease.

PREPARATIONS

Oral form

Clonidine hydrochloride tablets – 100 µg, 200 µg, 300 µg

Transdermal form

Delivering clonidine 100 µg, 200 µg or 300 µg daily for 1 week

Parenteral form

Clonidine hydrochloride 150 µg /ml ampoule

ASSESSMENT OF PAIN AND SEDATION

Pain is a complex, multidimensional symptom resulting from a combination of tissue damage and nociception, previous pain experience, personal beliefs, culture and mood. Acute pain is relatively straightforward to assess as, unlike chronic pain, it generally bears a predictable relationship to obvious tissue damage. Because the level of postoperative pain tends to change rapidly throughout the postoperative course, especially early after surgery, a policy of regular assessment of pain using simple measurement tools is the best way to ensure that pain treatment can be appropriately titrated. Pain is considered as the "fifth vital sign"[30].

Tools for Evaluation of Pain[30]

To assess the severity of pain and the success of treatment, some form of quantitation of pain is necessary. Many pain scoring systems are available.

a) Categorical: A four or five point scale grading the pain as none, mild, moderate, severe and excruciating etc. This scale lacks sensitivity, but it has the advantage of simplicity.

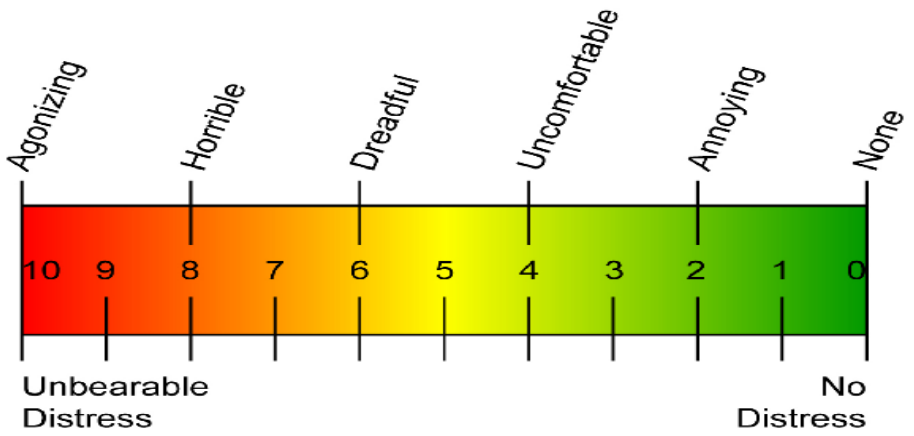
b) Numerical scale: This is an 11 point scale where “0” means no pain and “10” is the worst imaginable pain.

c) Visual Analogue Scale (VAS): A 10 cm scale with no pain at one end and worst imaginable pain at the other, is commonly used.

d) McGill Pain Questionnaire (MPQ): MPQ measures the sensory, affective, evaluative and other miscellaneous aspects of pain, thus measuring pain multi-dimensionally. The questionnaire contains about 20 aspects. 1 to 10 represents sensory aspects of pain, 11 to 15 represent affective aspect of pain, 16 represents evaluative aspect of pain, 17 to 20 other miscellaneous aspects of pain. Each subunit has 2 to 5 words under them, representing increasing degree of pain and numerical value. The sum of all points gives a rank value, which is termed the Pain Rating Index.

e) Happy-sad face – A child or an illiterate person could use a set of faces to indicate the severity of his pain. This scale shows a child’s face in different moods, the child is asked to select the facial expression that best suites the pain expression. This assesses the affective and fear component of pain.

In the study, pain was assessed using Visual Analogue Scale.



Preemptive Analgesia

Preemptive analgesia is described by Igor Kissin as "an antinociceptive therapy that prevents establishment of altered processing of afferent input, which amplifies postoperative pain." In simpler terms, a preemptive analgesic intervention is one that stops or alters pain transmission so that pain will not become amplified by the nervous system. Kissin's definition embraces the fundamental principles necessary for understanding the concept of preemptive analgesia: (a) that the central nervous system is capable of changing so that pain becomes either improved or worsened via central processes such as desensitization and sensitization, and (b) that alterations in sensory and pain transmission can effect such changes. Pain treated early in its course is easier to control than established pain. Several preemptive treatments—epidural analgesia, NSAIDs, nerve blocks and wound infiltration — are strongly effective.

Using the broader definition of preemptive analgesia that covers incisional (intraoperative) and inflammatory (postoperative) injury, the combination of experimental data and positive clinical trials strongly suggests that preemptive analgesia is a clinically relevant phenomenon. Maximal clinical benefit is observed when there is complete blockade of noxious stimuli with extension of this block into the postoperative period. By preventing central sensitization, preemptive analgesia along with intensive multimodal analgesic interventions could theoretically reduce acute postoperative pain/hyperalgesia and chronic pain after surgery.

Sedation scoring

The degree of sedation was evaluated by using the University of Michigan Sedation Scale (UMSS)[8] of 0 to 4

0 = awake and alert

1 = minimally sedated/sleepy, appropriate response to conversion and/or sound

2 = moderately sedated, somnolent/sleepy, easily aroused with tactile stimulation and/or simple verbal command

3 = deeply sedated/deep sleep, aroused only with significant stimulation

4 = could not be aroused

REVIEW OF LITERATURE

G. Andrieu et al [1] studied the analgesic efficacy of bilateral superficial cervical plexus block performed under general anaesthesia in patients undergoing total thyroidectomy, using ropivacaine or ropivacaine plus clonidine 5 µg/ml. During surgery, the median sufentanil requirements were significantly reduced in ropivacaine plus clonidine group. After surgery, the number of patients requiring nefopam within 24 h of surgery was significantly lower in these groups than in placebo group. At post-anaesthetic care unit admission, median pain scores were also significantly lower.

Rita Pal et al [2] studied the quality and duration of postoperative analgesia by cervical plexus block using bupivacaine and clonidine. Duration of analgesia was significantly more in bupivacaine plus clonidine group (8.19 ± 3.2 hours) as compared to bupivacaine group (5.24 ±1.6 hours). Total consumption of fentanyl citrate in postoperative period was also significantly less in the bupivacaine plus clonidine group.

Sophie Aunac et al [3] evaluated the analgesic efficacy of combined deep and superficial cervical plexus block in patients undergoing thyroidectomy under general anaesthesia. Bilateral combined deep and

superficial cervical block was given with saline (Group 1), ropivacaine (Group 2), or ropivacaine plus clonidine 7.5 μg/mL (Group 3). During surgery, the number of additional alfentanil boluses was significantly reduced in Groups 2 and 3 compared with Group 1. After surgery, the opioid and non-opioid analgesic requirements were also significantly reduced in Groups 2 and 3 during the first 24 h.

Danelli G et al [4] evaluated the effects of adding 50 μg clonidine to 150 mg ropivacaine for superficial cervical plexus block in patients undergoing elective carotid endarterectomy. They concluded that it shortened the onset time and improved the quality of surgical anaesthesia in patients undergoing elective carotid endarterectomy.

Ming-Lang Shih et al [5] investigated the analgesic efficacy of bilateral superficial cervical plexus block in patients undergoing thyroidectomy and to determine whether it reduces the adverse effects of general anaesthesia. They concluded that bilateral superficial cervical plexus block reduces general anaesthetics required during thyroidectomy. It also significantly lowers the severity of postoperative pain during the first 24 h and shortens the hospital stay.

Nathalie Dieudonne et al [6]studied the analgesic efficacy of bilateral superficial cervical plexus blocks performed at the end of thyroid surgery

using 20 mL isotonic sodium chloride or 20 mL bupivacaine 0.25% with 1:200,000 epinephrine. The Bupivacaine group had a smaller proportion of patients given morphine post operatively and lower initial median pain scores. It was concluded that bilateral superficial cervical plexus blocks significantly reduce pain intensity in the postoperative period after thyroid surgery but do not provide optimal pain relief alone.

Walter Pinto Neto et al [7] compared the analgesic effects of clonidine associated with bupivacaine to those of bupivacaine in cervical plexus block for carotid endarterectomy. They concluded that the association of 150 μg of clonidine and bupivacaine in cervical plexus block for carotid endarterectomy did not improve significantly the analgesic effects evaluated by pain severity, time until the first analgesic supplementations and amount of supplementary analgesics.

Zeynep Eti et al[10] compared the analgesic efficacy of bilateral superficial cervical plexus block and local anesthetic wound infiltration after thyroid surgery. They concluded that bilateral superficial cervical plexus block or local anesthetic wound infiltration with 0.25% bupivacaine did not decrease analgesic requirement after thyroid surgery.

Rowan R. Molnar et al[11] compared the addition of clonidine 5 μg/mL to lidocaine 1.5% for cervical plexus block with the addition of

epinephrine 5 μg/mL for carotid endarterectomy. The block onset time and duration were not different between the two groups. During the period from completion of the block until incision there was a significant heart rate increase in the epinephrine group as compared with the clonidine group. They concluded that clonidine 5 μg/mL is a useful additive to lidocaine 1.5% for cervical plexus block to reduce the incidence of tachycardia; however, omission of epinephrine results in higher serum lidocaine levels.

Susmita Chakraborty et al[8] evaluated the effect of clonidine as adjuvant in bupivacaine-induced supraclavicular brachial plexus block. They concluded that addition of a small dose of clonidine to 0.5% bupivacaine significantly prolonged the duration of analgesia without producing any clinically important adverse reactions other than sedation.

Hutschala, D et al[9] demonstrated that the admixture of clonidine to bupivacaine plus epinephrine prolongs and enhances brachial plexus blockade. Lower clonidine plasma concentrations for block treatment strongly suggested a local effect.

McCartney CJ et al[12] showed that Clonidine improves the duration of analgesia and anaesthesia when used as an adjunct to intermediate-acting local anaesthetics for peripheral nerve blocks. They observed that

side-effects appear to be limited at doses up to 150 μg, but found that evidence is lacking for the use of clonidine as an adjunct to local anaesthetics for continuous catheter techniques.

Giovanni Cucchiaro et al [13] evaluated the effects of clonidine on the duration of sensory and motor block and analgesia time in children who underwent a variety of peripheral nerve blocks. The patients received either bupivacaine or ropivacaine local anesthetic (LA) and a combination of local anesthetic and clonidine (LAC). The duration of sensory block was significantly longer in the LAC (17.2 ± 5 h) compared with that in the LA group (13.2 ± 5 h). The increase in duration was independent from the type of peripheral nerve block, local anesthetic used and operation performed.

Andrea Casati et al [14] demonstrated that, when providing combined sciatic-femoral nerve block for hallux valgus repair, the addition of 1 μg/kg clonidine to 0.75% ropivacaine prolongs the duration of postoperative analgesia by 3 h, with only a slight and short-lived increase in the degree of sedation and no hemodynamic adverse effects.

Aruna Parameswari et al [15] demonstrated that clonidine in a dose of 1 μg/kg added to 0.25% bupivacaine for caudal analgesia, during sub-umbilical surgeries, prolongs the duration of analgesia of bupivacaine,

without any side effects. 0.25% bupivacaine group had a mean duration of analgesia of 288.7 ± 259.1 min and 0.25% bupivacaine with 1 μg/kg of clonidine added group had a mean duration of analgesia of 593.4 ± 423.3 min.

H. El Saied et al [16]demonstrated that addition of 150 μg of clonidine to ropivacaine, for brachial plexus blockade, prolongs motor and sensory block and analgesia, without an increased incidence of side effects.

Brian D Sites et al [17] studied the effects of intrathecal clonidine for total knee arthroplasty using a hyperbaric bupivacaine spinal anesthetic. It was concluded that the co-administration of intrathecal clonidine and morphine decreased the 24-h IV morphine consumption and improves the 24-h VAS score when compared with intrathecal morphine alone.

Scott S. Reuben et al [18] studied the effect of clonidine as a component of intravenous regional anaesthesia. They concluded that the addition of 1 μg/kg clonidine to lidocaine, 0.5%, for IVRA in patients undergoing ambulatory hand surgery improves postoperative analgesia without causing significant side effects during the first postoperative day.

Jean-Marc Bernard et al [19] studied the dose-Range Effects of Clonidine Added to Lidocaine for Brachial Plexus Block. 45 ml of a

mixture containing either 400 mg lidocaine plus saline or 400 mg lidocaine plus 30, 90 or 300 μg clonidine was used for axillary nerve block. Their study showed that 30 μg clonidine was more effective than 90 μg clonidine in producing sensory blockade. Sedation occurred with clonidine 30 and 300 μg. Clonidine reduced the use of supplementary intravenous anesthetic agents for surgery and produced dose-dependent prolongation of analgesia, reaching a mean 770 min (range, 190-1440 min) for the largest dose.

DJ Reinhart et al [20] demonstrated that compared to 1.73% lidocaine, combining clonidine (10 μg /mL) with lidocaine for local anesthetic block for foot surgery significantly increases the duration and quality of postoperative analgesia.

MATERIALS AND METHODS

STUDY DESIGN AND EQUIPMENT

This was a Prospective Randomized Double Blind study conducted in 40 adult patients undergoing thyroidectomy surgeries from August 2009 to September 2010 under General Anaesthesia at Chengalpattu Medical College Hospital, Chengalpattu.

The study was done with Ethical Committee approval and written informed consent obtained from all patients included in the study.

An Anaesthesiology Consultant who did not take any part in the study drew the group B or group C drug in a syringe and coded them and was handed over to an Anaesthesiology resident who performed the block. Another resident was asked to conduct the anaesthesia and also take down the data as per the proforma attached.

SELECTION OF CASES

Inclusion Criteria

- ASA grade I & II patients of either sex
- Age 20 – 60 yrs
- Euthyroid patients
- Type of surgery: Thyroidectomy

Exclusion criteria

- Substernal goiters
- Thyroidectomy with lymph node dissection
- Inability to use the pain scale
- Coagulation disorders

METHOD

40 patients under ASA I and II scheduled to undergo elective thyroidectomy surgery were included in this study.

Patients included in the study underwent thorough preoperative evaluation which included detailed history, physical examination, investigations (Hemoglobin, PCV, Blood Urea, Serum Creatinine, Serum Electrolytes, Random blood sugar, Thyroid Function Test, Urine albumin-sugar, ECG, Chest X Ray, X Ray Neck) and Indirect Laryngoscopy.

The patients were randomly allocated into two groups – Group B and Group C

All the patients were premedicated with oral ranitidine 150mg two hours before surgery with a sip of water. Anaesthesia machine, drug trolley including emergency drugs and difficult airway tray were checked.

Patients were monitored with Non invasive blood pressure monitoring, Pulse oximeter, five lead ECG and temperature.

Intraoperatively patients in both groups received analgesia with Inj.Fentanyl Citrate 2µg/kg, 5 minutes before induction. Inj. Xylocard 1.5mg/kg was given 90s before induction. Induction of anaesthesia was done with Inj.Propofol 2mg/kg. Intubation with Inj.Atracurium 0.5mg/kg.

Maintanence with titrated Propofol infusion, nitrous oxide/ oxygen with top up doses of Atracurium and 0.5 µg/kg of Inj.Fentanyl Citrate topped up every hour.

After intubation, bilateral superficial cervical plexus block was performed. The patient in the supine position and the head and neck turned opposite the side to be blocked. The superficial cervical plexus was blocked at the midpoint of the posterior border of the sternocleidomastoid muscle. A 24-gauge, 2.5cm needle is inserted subcutaneously posterior to the sternocleidomastoid muscle, and 2 mL of local anesthetic was injected. The needle is redirected both superiorly and inferiorly along the posterior border of the sternocleidomastoid, and 2 mL of solution was injected along each of these sites. A total of 6 mL of local anaesthetic was thus injected

Group B was assigned as 6ml of 0.25% Bupivacaine on each side

Group C was assigned as 6ml of 0.25% Bupivacaine with Clonidine 5µg/ml on each side

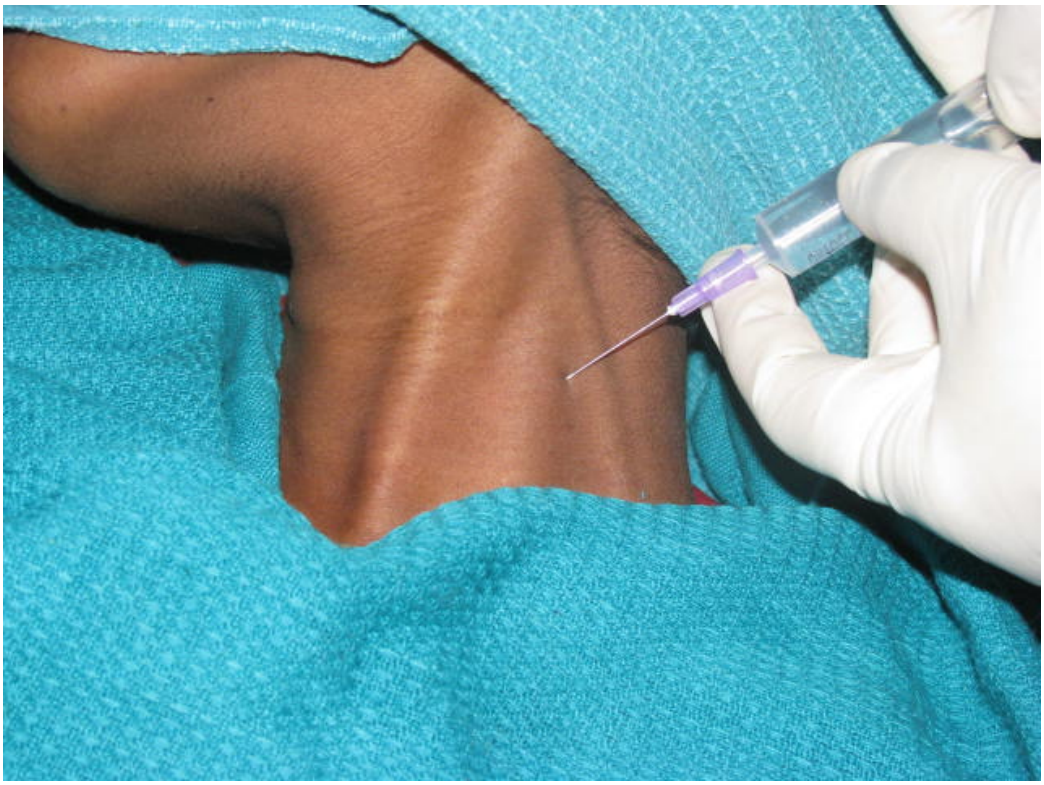
At the end of the surgery patient extubated following adequate neuromuscular reversal with Neostigmine 0.05mg/kg and Glycopyrrolate 0.01mg/kg, intensity of pain assessed by visual analogue scale (VAS) and sent to PACU.

The patients were assessed intraoperatively and postoperatively till VAS Score was 3 as per our proforma.

Physiological measures assessed intraoperatively were heart rate, SpO_2 and blood pressure at 0,5,10,15,20,25,30 minutes and every 15 minutes there after till the end of surgery.

Postoperatively, heart rate, blood pressure, sedation score as per the University of Michigan Sedation Scale and pain by visual analogue scale (VAS) were recorded every 30 minutes for the first 6 hrs, then every hour till the VAS score was 3. Rescue analgesic with Inj Fentanyl citrate 2µg/kg was given when VAS was 3 and they were taken out of my study.

Duration of effective analgesia was defined as the time interval from the administration of block to the time to reach VAS score of 3. Patients were monitored for any hypotension, bradycardia and excessive sedation.



Method of needle placement for a superficial cervical plexus block

OBSERVATIONS & RESULTS

Statistical Analysis

All collected data were entered into computer using MS Excel software and analysed using STATA software. Descriptive analysis presented in the form of Tables and Graphs. The level of significance was 0.05 used for determining the significance of different variables. Student 't' test was used to determine the significance of quantitative variables (heart rate, systolic, diastolic and mean arterial pressure). Chi-square exact test was used to test the significance of qualitative data (pain and sedation score).

DEMOGRAPHICS

1. Distribution Of Mean Age By Groups

Parameters	Group B	Group C	p-value
No. of cases	20	20	0.7
Mean	37.6	36.2	

2. Distribution Of Mean Weight (Kg) By Groups

Parameters	Group B	Group C	p-value
No. of cases	20	20	0.17
Mean	55	53	

The groups were comparable with respect to their age and weight.

3. Duration Of Surgery

Parameters	Group B	Group C	p-value
No. of cases	20	20	0.51
Mean	123.15	128.25	
Standard deviation	23.2	24.1	

There is no statistically significant difference between the two groups with respect to the duration of surgery.

4. Distribution of mean, standard deviation and significance between two groups at base recording

Group (Nos.)	SBP		DBP		MAP		HR	
	Mean	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**
B (20)	124	6.6	81.5	5.1	95.7	5.4	84.4	3
C (20)	124.6	6.6	82.5	4.1	96.5	4.5	85.9	6.3
probability	P=0.77		P=0.47		P=0.6		P=0.32	

5. Distribution of mean, standard deviation and significance between two groups at 0th minute recording of intra operative period

Group (Nos.)	SBP		DBP		MAP		HR	
	Mean	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**
B (20)	128	6.6	83.7	5.8	98.4	5.7	86.7	2.7
C (20)	127.6	9.1	85.4	5.6	99.1	6.97	89.8	10.5
probability	0.87		0.35		0.73		0.21	

6. Distribution of mean, standard deviation and significance between two groups at 15th minute recording of intra operative period

Group (Nos.)	SBP		DBP		MAP		HR	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
B (20)	113.2	7.4	73.2	4.0	86.6	4.6	77.8	4.9
C (20)	109.6	11.3	70.4	6.4	83.5	7.3	77.2	5.5
probability	0.24		0.10		0.12		0.72	

7. Distribution of mean, standard deviation and significance between two groups at 30th minute recording of intra operative period

Group (Nos.)	SBP		DBP		MAP		HR	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
B (20)	117.5	8.3	76.3	7.7	90.1	7.5	79	5.1
C (20)	115.9	8	76.1	5.8	89.3	6.0	77.1	4.2
probability	0.52		0.92		0.71		0.61	

8. Distribution of mean, standard deviation and significance between two groups at 45th minute recording of intra operative period

Group (Nos.)	SBP		DBP		MAP		HR	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
B (20)	118	7.1	77.3	6.5	90.8	6.2	80.2	4.9
C (20)	116.5	8.7	75.8	5.9	89.5	6.4	79.3	6.3
probability	0.59		0.43		0.51		0.62	

9. Distribution of Mean, Standard deviation and significance between two groups at 60th minute recording of intra operative period

Group(No)	SBP		DBP		MAP		HR	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
B (20)	120	5.2	78.6	6	92.4	5.2	79.9	5.5
C (20)	119.4	8.9	78.4	6.5	92	6.8	79.6	5.9
probability	0.83		0.90		0.85		0.87	

10. Distribution of Mean, Standard deviation and significance between two groups at 90th minute recording of intra operative period

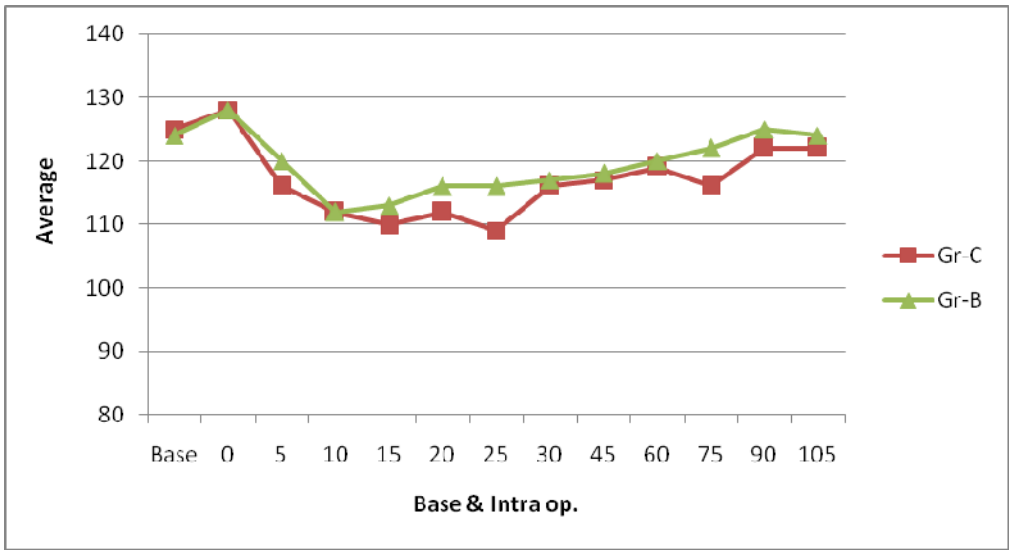
Group (Nos.)	SBP		DBP		MAP		HR	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
B (20)	124.5	6.7	82.2	5.6	96.3	5.6	82	5.9
C (20)	121.8	6.1	79.1	5.5	93.3	5.1	81.1	7.6
probability	0.18		0.08		0.08		0.67	

11. Distribution of Mean, Standard deviation and significance between two groups at 105th minute recording of intra operative period

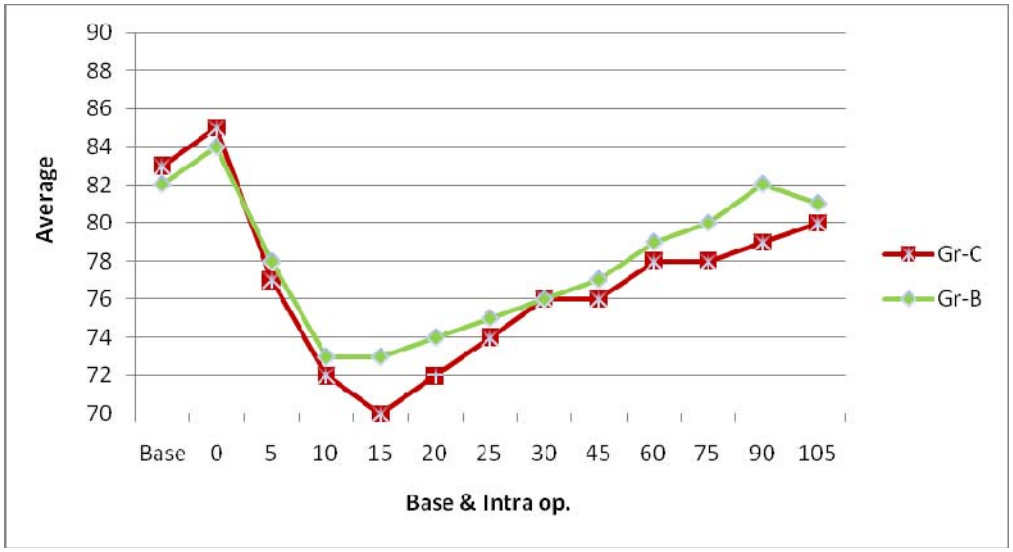
Group (Nos.)	SBP		DBP		MAP		HR	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
B (17)	123.7	6.7	81	6.3	95.3	6.1	83	3.9
C (16)	122.4	5.8	79.7	6.3	94	5.7	81.8	7.7
probability	0.53		0.57		0.55		0.56	

There is **no statistically significant difference** between two groups of patients on systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP) and heart rate (HR) in the intraoperative period.

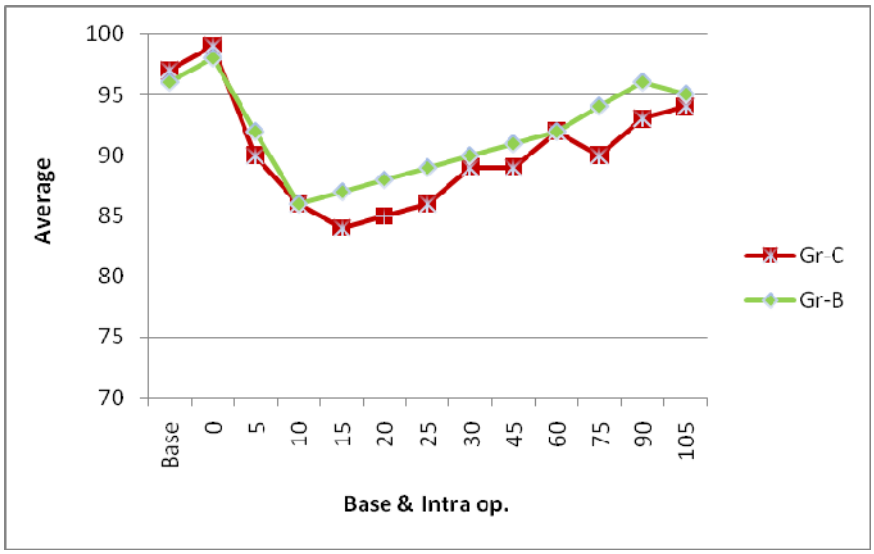
Distribution of SBP over base and intra operative period



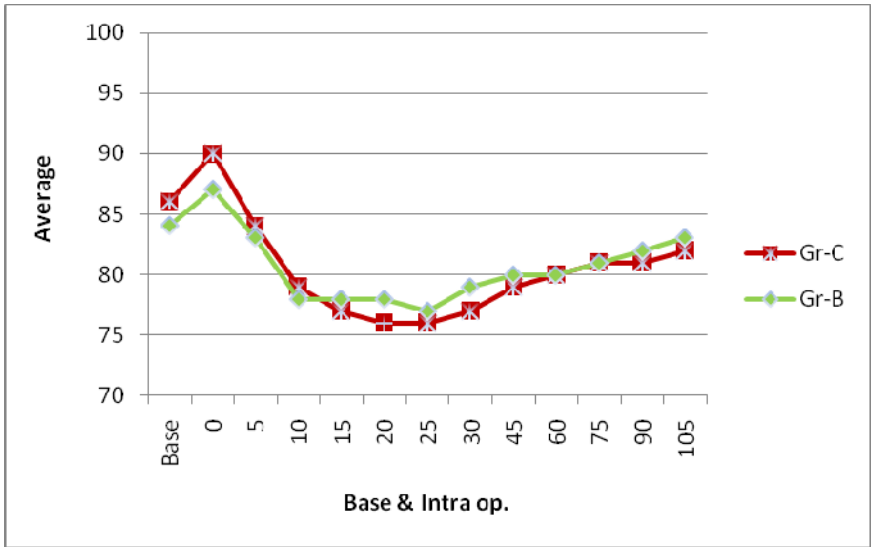
Distribution of DBP over base and intra operative period



Distribution of MAP over base and intra operative period



Distribution of HR over base and intra operative period



12. Distribution of Mean, Standard deviation and significance between two groups at 0th hour recording of post operative period

Group (Nos.)	SBP		DBP		MAP		HR	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
B (20)	123.6	4.1	82.7	2.5	96.4	2.5	84.5	4.2
C (20)	123.7	6.3	80.8	5.8	95.2	5.5	84.4	4.7
Probability	0.95		0.19		0.38		0.91	

13. Distribution of Mean, Standard deviation and significance between two groups at 0.5th hour recording of post operative period

Group (Nos.)	SBP		DBP		MAP		HR	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
B (20)	121	4.4	79.4	5.2	93.3	4.4	83	3.2
C (20)	120.5	6.6	78.2	6.5	92.3	6.2	82.7	4
probability	0.80		0.50		0.53		0.82	

14. Distribution of Mean, Standard deviation and significance between two groups at 1st hour recording of post operative period

Group (Nos.)	SBP		DBP		MAP		HR	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
B (20)	121.7	5.2	79.7	4.8	93.8	4.5	81.7	3.9
C (20)	120.3	6.3	78.9	4.8	92.7	4.6	81.3	3.7
probability	0.44		0.57		0.42		0.74	

15. Distribution of Mean, Standard deviation and significance between two groups at 1.5th hour recording of post operative period

Group (Nos.)	SBP		DBP		MAP		HR	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
B (20)	123.1	4.9	80.2	4.6	94.5	4.2	81.4	3.4
C (20)	119.7	6.6	79.3	5.7	92.7	5.3	79.8	3.5
probability	0.08		0.58		0.26		0.15	

16. Distribution of Mean, Standard deviation and significance between two groups at 2nd hour recording of post operative period

Group (Nos.)	SBP		DBP		MAP		HR	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
B (19)	122.7	5.0	80.4	4.9	94.6	4.6	80.8	4.3
C (20)	119.9	5.5	78.2	5.4	92.1	4.9	79.6	3.9
probability	0.09		0.18		0.10		0.31	

17. Distribution of Mean, Standard deviation and significance between two groups at 2.5th hour recording of post operative period

Group (Nos.)	SBP		DBP		MAP		HR	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
B (19)	117.6	21.3	80.3	5.2	92.6	7.6	80.7	3.8
C (20)	120.2	5.5	77.4	5.3	91.6	5	79.1	3.7
probability	0.59		0.097		0.60		0.18	

18. Distribution of Mean, Standard deviation and significance between two groups at 3rd hour recording of post operative period

Group (Nos.)	SBP		DBP		MAP		HR	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
B (19)	123.8	5.2	80.7	4.7	95	4.7	81.3	3.7
C (20)	120.9	5.5	78.3	5.2	92.5	4.7	78.9	3.8
probability	0.094		0.14		0.10		0.060	

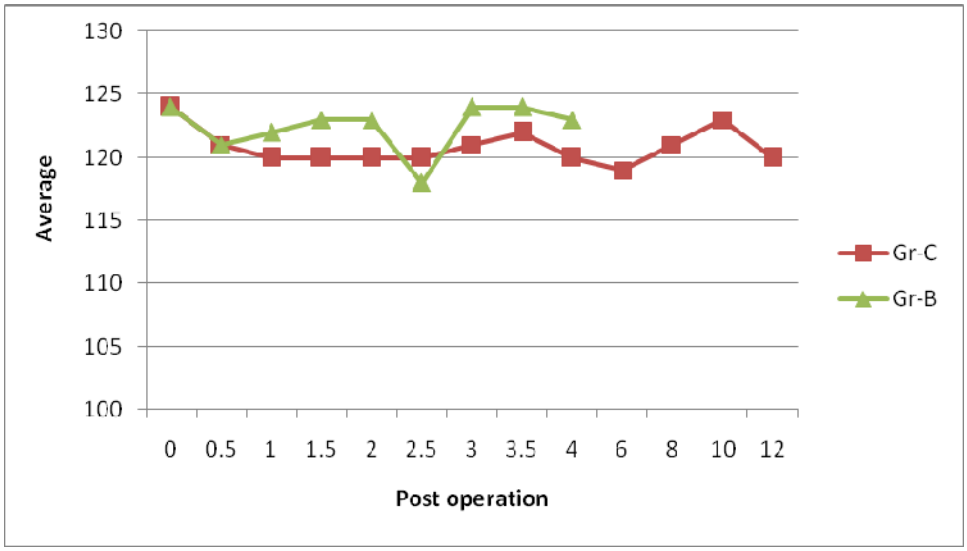
19. Distribution of Mean, Standard deviation and significance between two groups at 4th hour recording of post operative period

Group (Nos.)	SBP		DBP		MAP		HR	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
B (12)	122.9	3.6	80.5	4.7	94.6	4.4	81.2	4.2
C (20)	119.6	5	77.4	5	91.5	4.3	79.8	4.6
probability	0.06		0.08		0.12		0.06	

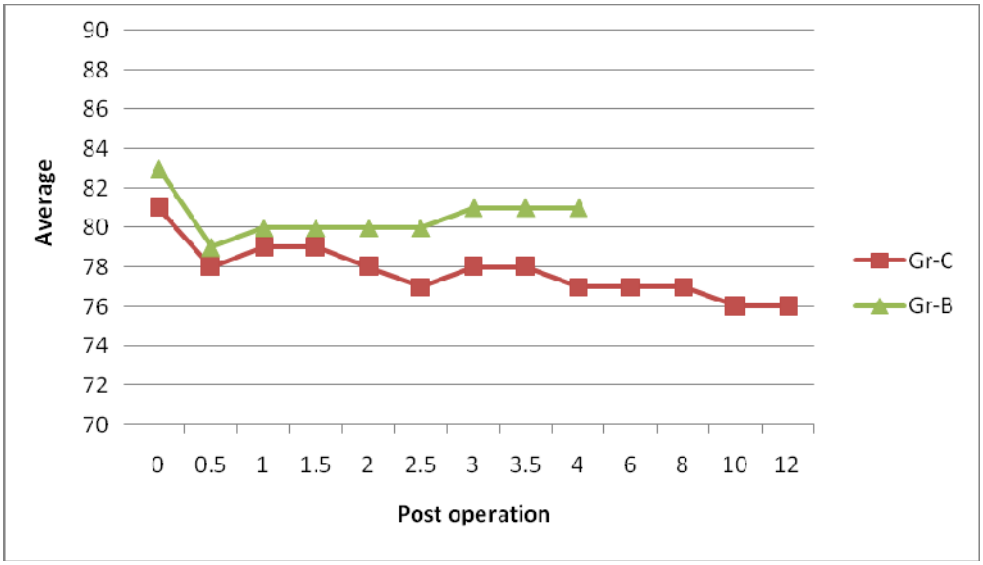
There is **no statistically significant difference** between two groups of patients on systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP) and heart rate (HR) in the postoperative period.

In the fifth hour there were only 3 patients in group B. In the sixth and seventh hours, there was only one patient in group B. Beyond this period there are no patients in group B.

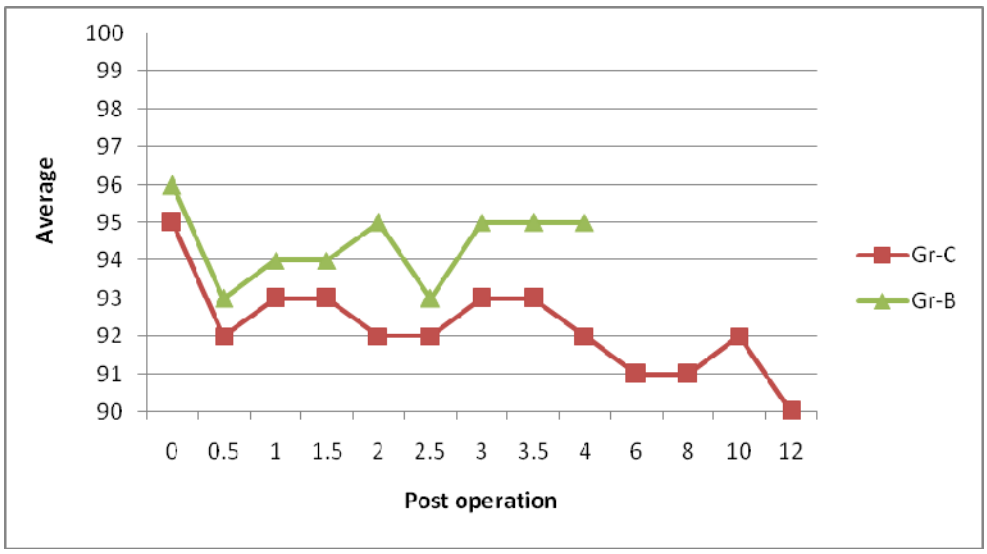
Distribution of SBP in Post operative period



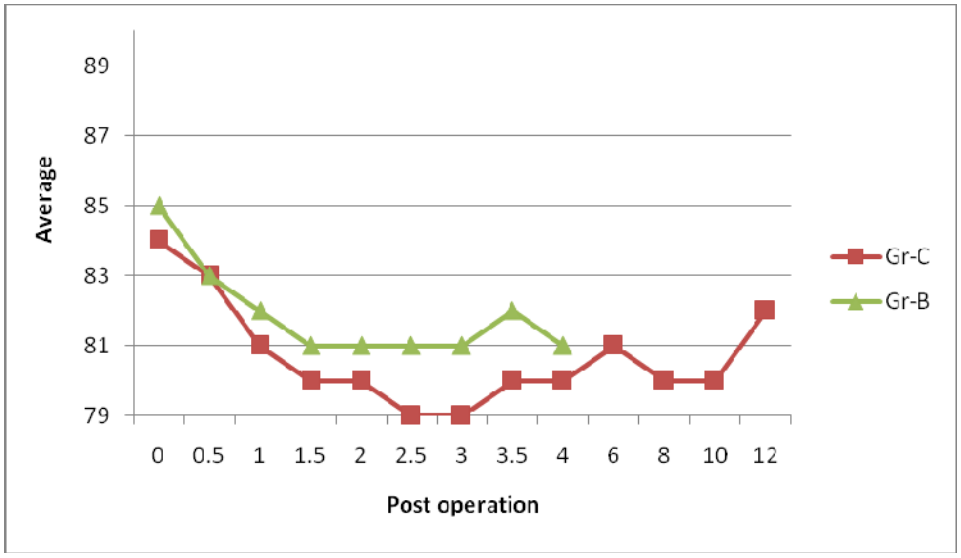
Distribution of DBP in Post operative period



Distribution of MAP in Post operative period



Distribution of HR in Post operative period



20. Comparison of VAS in the post operative period

VAS at 0th postoperative hour

Group (No)	VAS	
	0	**1**
B (20)	20	0
C (20)	20	0

All the patients in both the groups had a VAS of 0 at 0th postoperative hour.

VAS at 0.5th postoperative hour

Group (No)	VAS	
	0	**1**
B (20)	19	1
C (20)	20	0

All the patients (100%) in group C had a VAS of 0 and 19 patients (95%) in group B had a VAS of 0. Only 1 patient (5%) in group B had a VAS of 1.

VAS at 1st postoperative hour

Group (No)	VAS		
	0	**1**	**2**
B (20)	7	12	1
C (20)	15	5	0

Chi square = 6.7, p = 0.034

In Group C, 75% patients had a VAS of 0, 25% had a VAS of 1.

In Group B, 35% patients had a VAS of 0, 60% patients had a VAS of 1 and 5% patients had a VAS of 2. This is **statistically significant (p = 0.034)**

VAS at 1.5th postoperative hour

Group (No)	VAS		
	0	**1**	**2**
B (20)	1	18	1
C (20)	9	11	0

Chi square = 9.1, p = 0.011

In Group C, 45% patients had a VAS of 0, 55% had a VAS of 1.

In Group B, 5% patients had a VAS of 0, 90% patients had a VAS of 1 and 5% patients had a VAS of 2. This is **statistically significant (p = 0.011)**

VAS at 2nd postoperative hour

Group (No)	VAS		
	0	**1**	**2**
B (19)	0	11	8
C (20)	3	17	0

Chi square = 12.26, p = 0.002

In Group C, 15% patients had a VAS of 0, 85% had a VAS of 1.

In Group B, 58% patients had a VAS of 1 and 42% patients had a VAS of 2. This is **statistically significant (p = 0.002).**

VAS at 2.5th postoperative hour

Group (No)	VAS		
	0	**1**	**2**
B (19)	0	6	13
C (20)	1	19	0

Chi square = 20.7, p = 0.0001

In Group C, 5% patients had a VAS of 0, 95% had a VAS of 1.

In Group B, 32% patients had a VAS of 1 and 68% patients had a VAS of 2. This is **statistically significant (p = 0.0001)**

VAS at 3rd postoperative hour

Group (No)	VAS			
	0	**1**	**2**	**3**
B (19)	0	1	16	2
C (20)	0	19	1	0

In Group C, 95% patients had a VAS of 1and 5% had a VAS of 2.

In Group B, 5% patients had a VAS of 1 and 84% patients had a VAS of 2 and 11% had a VAS of 3.

VAS at 4th postoperative hour

Group (No)	VAS			
	0	**1**	**2**	**3**
B (12)	0	0	6	6
C (20)	0	13	7	0

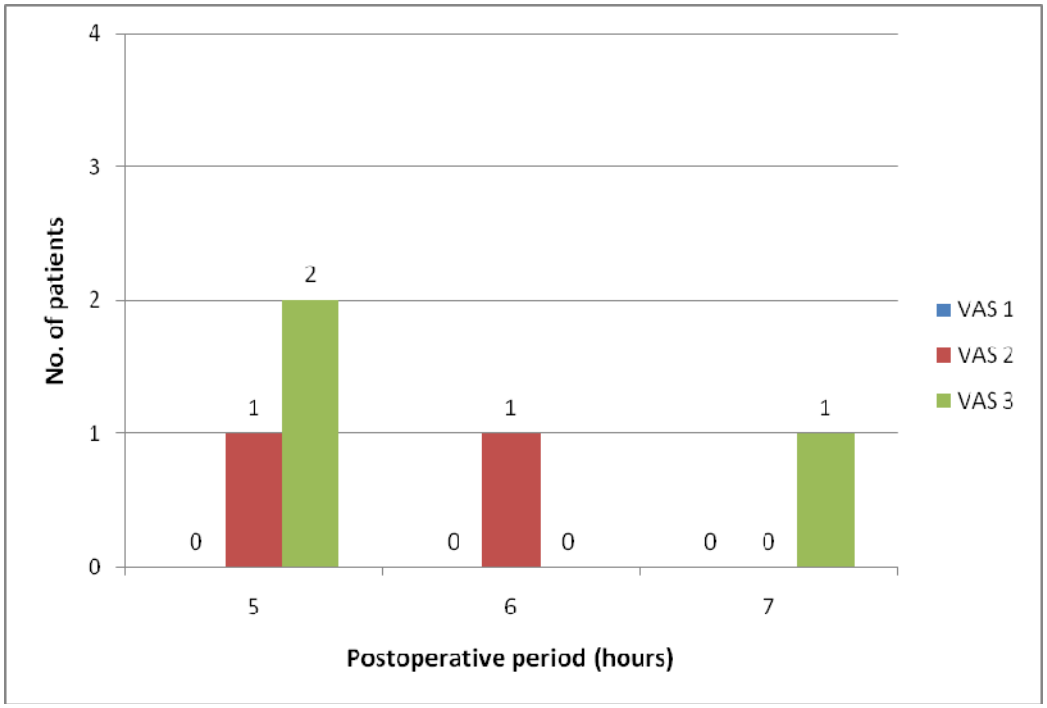
In Group C, 65% patients had a VAS of 1and 35% had a VAS of 2.

In Group B, 50% patients had a VAS of 2 and 50% patients had a VAS of 3.

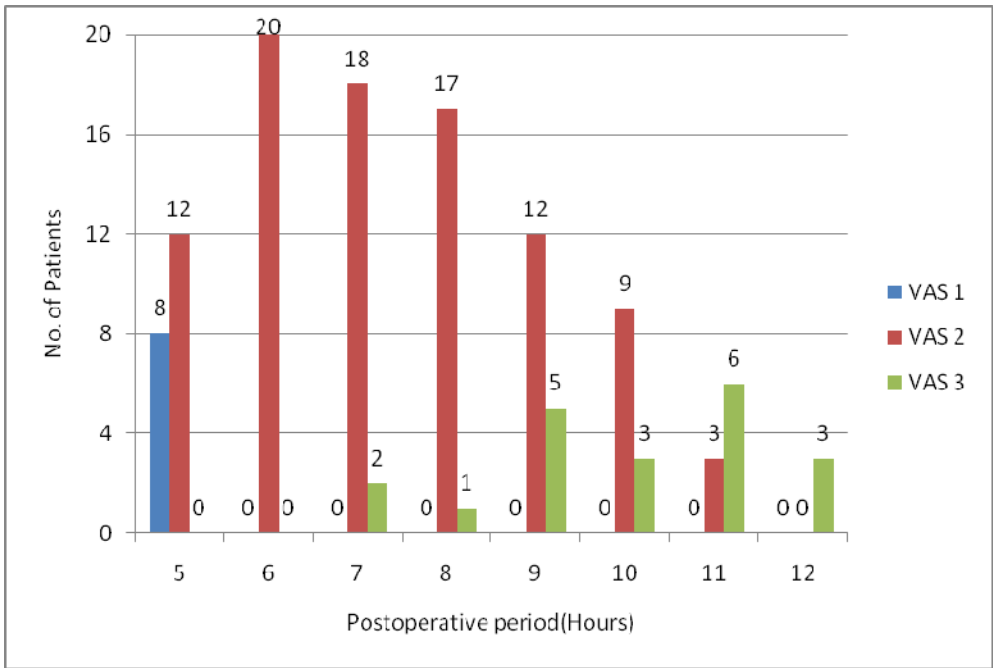
In the **5th postoperative hour**, there are only 3 patients in group B, of which 1 patient had a VAS score of 2. 2 patients had a VAS score of 3 and they were given rescue analgesic and were out of my study.

In the **6th postoperative hour**, there is only 1patient in group B who had a VAS score of 2. In the **7th postoperative hour**, there is only one patient in group B who had a VAS score of 3.

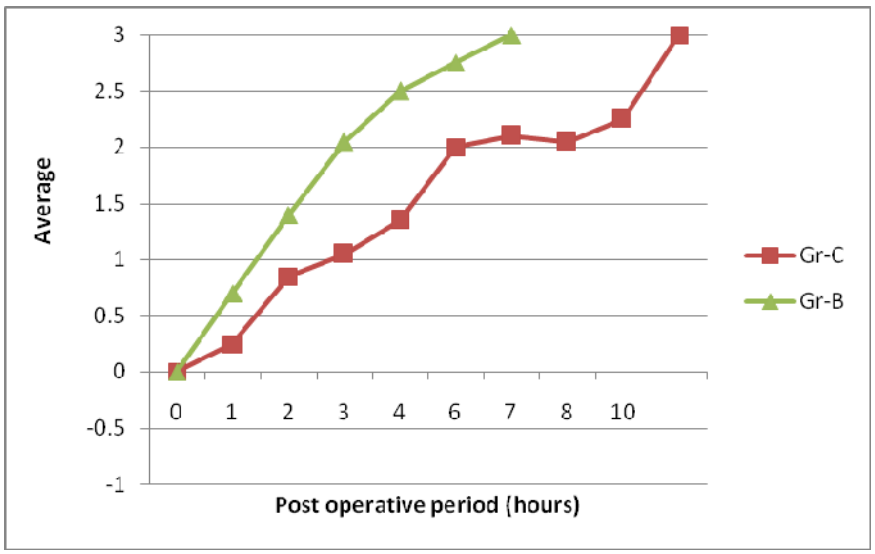
The VAS score distribution of the number of patients in **group B** from 5th hour is shown in the following graph.



The VAS score distribution of the number of patients in **group C** from 5th hour is shown in the following graph.



Average VAS score distribution over different hours of post operative period



21. Comparison of sedation score in the postoperative period

Sedation Score at 0th postoperative hour

Group	Sedation Score	
	0	**1**
B	18	2
C	0	20

In Group B, 90% patients had a sedation score of 0 and 20% had a sedation score of 1.

In Group C 100% patients had a sedation score of 1.

Sedation Score at 0.5th postoperative hour

Group	Sedation Score	
	0	**1**
B	20	0
C	3	17

Chi square = 29.5, p=0.00001

In Group B, 100% patients had a sedation score of 0.

In Group C 85% patients had a sedation score of 1 and 15% had a sedation score of 0. This is **statistically significant (p = 0.00001)**

Sedation Score at 1st postoperative hour

Group	Sedation Score	
	0	**1**
B	20	0
C	14	6

Chi square = 7.1, p = 0.011

In Group B, 100% patients had a sedation score of 0.

In Group C 30% patients had a sedation score of 1 and 70% had a sedation score of 0. **This is statistically significant (p = 0.011)**

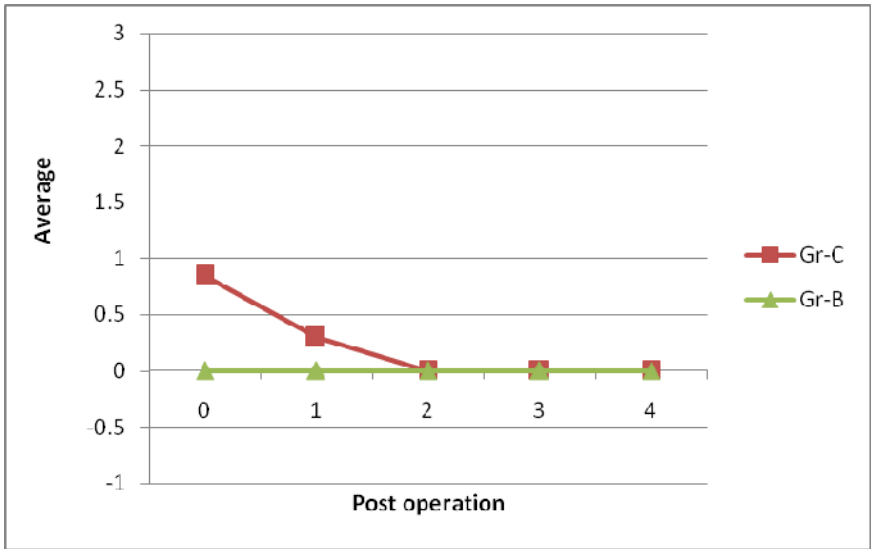
Sedation Score at 2nd postoperative hour

Group	Sedation Score	
	0	**1**
B	19	0
C	20	0

In both the groups all the patients had a sedation score of 0.

Beyond this period patients of both groups had a score of 0.

Sedation Score distribution over different hours of post operative period



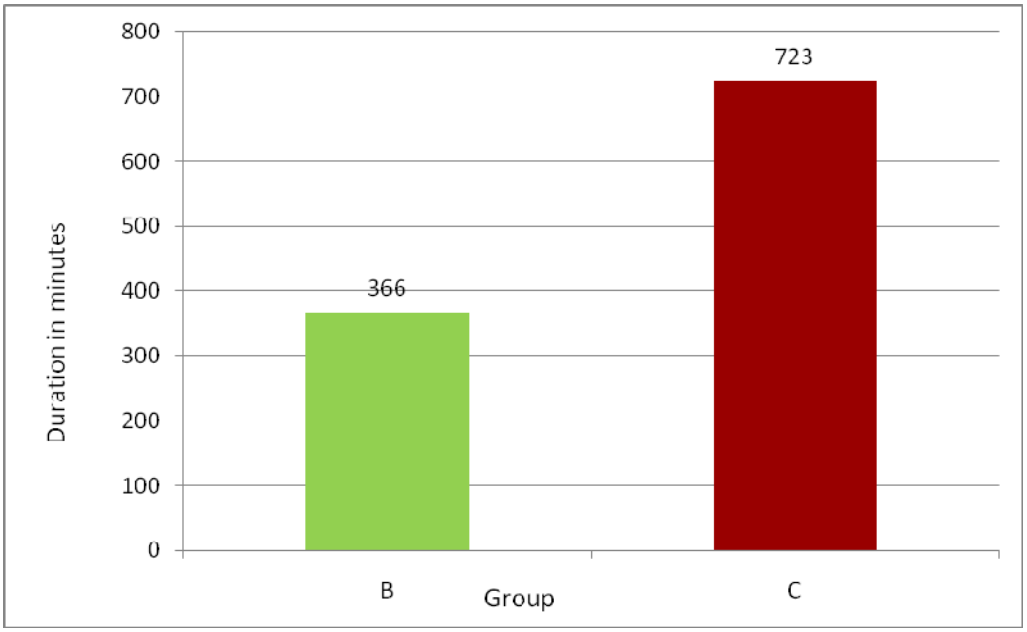
21. Duration of analgesia

Mean and Standard deviation of duration of analgesia and its significance in groups

Group	Mean (minutes)	Std. Deviation	P value
B	366	60.1	0.00001
C	723	94.6	

There is **highly significant difference** between two groups in the duration of analgesia.

Duration of analgesia



DISCUSSION

Pain after thyroid surgery is regarded as being of moderate intensity. During the first 24 h after surgery, patients require opioid and non-opioid analgesics. Addition of a regional anaesthesia to general anaesthesia is an appropriate component of multimodal analgesia in this setting. Bilateral superficial cervical plexus block is found to reduce postoperative analgesic requirements after thyroid surgery.

Thyroidectomy surgery done only under combined superficial and deep cervical plexus block was associated with more serious complications particularly accidental injury into vertebral artery, phrenic nerve palsy and also it caused discomfort to the patient of being awake during surgery. Compared with bilateral deep cervical plexus block, bilateral superficial cervical plexus block has the advantage of being devoid of serious complications.

The present study was designed to evaluate the effectiveness of Clonidine as an adjuvant to Bupivacaine for superficial cervical plexus block. A total of 40 patients were randomized into two groups (B & C). Both the groups were monitored for haemodynamic parameters like heart

rate, systolic and diastolic blood pressure, mean arterial blood pressure, duration of postoperative analgesia and sedation.

The mechanism whereby clonidine prolongs the duration of local anaesthetic blockade when injected into a nerve sheath remains speculative. Clonidine is highly lipid soluble, easily crosses the blood brain barrier to interact with alpha-2 adrenergic receptors at both spinal and supraspinal sites within the central nervous system producing its analgesic effect. Epidural and intrathecal clonidine does produce analgesia when used as the sole analgesic agent. Clonidine alone injected into the nerve sheath failed to produce prolonged analgesia.

Clonidine injected into peripheral nerve sheath may act by translocation via nerves (axonal transport) or blood stream to the spinal cord as has become evident with opioid analgesics. The effect may occur by direct action of clonidine on nerve fibre conduction, specifically C and A delta fibres. However, this would require high local concentrations and would not explain why clonidine alone injected into the nerve sheath failed to produce prolonged analgesia. The action of clonidine would then more likely be via a synergistic mechanism of action in combination with the local anaesthetic resulting in the prolonged effect[16]. Clonidine possibly enhances or amplifies the sodium channel blockade action of

local anaesthetics by opening up the potassium channels resulting in membrane hyperpolarization, a state in which the cell is unresponsive to excitatory input[8]. This is probably the only mechanism that would explain the extended duration of both the sensory and motor blockade. What is certain is that clonidine has mixed alpha-1 and alpha-2 agonist effects at both pre and postsynaptic receptors as well as effects on a number of other specific receptors. Inhibition of noradrenaline release, mediated by an interaction with α2-adrenergic presynaptic receptors, could be an alternative explanation for the enhancing effect of the peripheral administration of clonidine[34]. Many authors favour the hypothesis that clonidine exerts its local anaesthetic-prolonging effect directly on the nerve fibre, as a result of complex interaction between clonidine and axonal ion channels or receptors[31,32,33]. Peripheral antinociception induced by clonidine has also been related to an α2-adrenoceptor mediated local release of enkephalin-like substance[35]. Its mechanism of action and effects, therefore, are likely to be compound and complex.

In our study, both the groups were comparable with respect to demographic details like age, weight of the patient and duration of surgery.

In the study, the postoperative analgesia was evaluated using the visual analog scale. Duration of effective analgesia was defined as the time interval from the administration of block to the time to reach VAS score of 3 and it was significantly higher in the clonidine group, with a p value of 0.00001. Mean duration of analgesia for the clonidine group was 723± 94.6 minutes compared to the mean duration of analgesia for the bupivacaine alone group of 366±60.1minutes.

The findings were consistent with the study of Rita Pal et al [2] who studied the quality and duration of postoperative analgesia by cervical plexus block using bupivacaine and clonidine. Duration of analgesia was significantly more in bupivacaine plus clonidine group (8.19 ± 3.2 hour) as compared to bupivacaine group (5.24 ±1.6 hour). It was also consistent with studies of Sophie Aunac et al[3] and Susmita Chakraborty et al[8] who observed the potentiation of analgesia by addition of clonidine.

In the postoperative period, patients administered clonidine were found to be sedated up to one hour. Sedation which was assessed by the University of Michigan Sedation Scale, never exceeded a score 1 (minimal sedation). Moreover, the addition of clonidine did not delay recovery from general anaesthesia. This was consistent with findings of

Susmita Chakraborty et al[8] who studied the effect of clonidine as an adjuvant in bupivacaine-induced supraclavicular brachial plexus block.

Hemodynamic parameters measured were heart rate, systolic, diastolic and mean arterial pressure. There is no significant difference between the two groups with respect to the systolic, diastolic and mean arterial pressure and heart rate in the intraoperative and postoperative period. This was consistent with findings of Susmita Chakraborty et al[8].

Though certain studies like that of Walter Pinto Neto et al [7] have found out that clonidine did not enhance local anesthetic effect in regional anaesthesia, our study substantiated its use as an adjunct to local anaesthetics to provide postoperative analgesia without deleterious side-effects.

CONCLUSION

Thyroidectomy is routinely done under general anaesthesia supplemented with postoperative narcotics in our institution. Thyroidectomy done under general anaesthesia and bilateral superficial cervical plexus block gives better patient comfort in the postoperative period. The addition of alpha-2 adrenergic agonist Clonidine as an adjuvant to local anaesthetic has improved and prolonged postoperative analgesia and mild sedation without any significant adverse effects. There is lesser consumption of postoperative narcotics and it also facilitates a smooth transition from intraoperative to postoperative period.

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PROFORMA

Name: Age/ Sex:

IP NO: WT:

Diagnosis: Surgery :

Duration of Surgery:

Preop Assessment:

Investigations:

Premedication:

Induction:

Intubation:

Maintanence:

Positioning:

Bilateral Superficial Cervical plexus block – drugs as per the random group B/C:

INTRAOPERATIVE

Time(min)	0	5	10	15	20	25	30	45	60	75	90	105	120	135	150	165	180
HR																	
SBP																	
DBP																	
MAP																	
SpO2																	
Atracurium																	
Fentanyl																	

POSTOPERATIVE

Time (HR)	0	0.5	1	1.5	2	2.5	3	3.5	4	4.5	5	5.5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24
VAS																															
HR																															
SBP																															
DBP																															
MAP																															
Sedation Score																															
Rescue analgesic																															

MASTER CHART

DATA OF PATIENTS - INTRAOP – BUPIVACAINE GROUP

S No	Name	Age	Sex	Wt	BHR	BSBP	BDBP	BMAP	0				5				10				15			
									HR	SBP	DBP	MAP	HR	SBP	DBP	MAP	HR	SBP	DBP	MAP	HR	SBP	DBP	MAP
1	Meena	26	F	53	90	120	84	96	92	126	86	99	88	120	82	95	86	108	69	82	84	112	72	85
2	Kala	60	F	50	82	134	86	102	86	138	92	107	84	130	84	99	79	132	82	99	82	124	76	92
3	Elumalai	60	M	60	80	130	82	98	84	136	88	104	88	130	85	100	82	122	79	93	80	120	74	89
4	Manjula	27	F	57	84	117	74	88	86	113	72	86	79	111	73	86	74	98	68	78	76	108	70	83
5	Mohana	30	F	60	84	122	81	95	88	130	86	101	82	122	84	97	79	101	69	80	83	103	72	82
6	Valarmathi	35	F	62	85	122	84	97	88	126	85	99	84	110	71	84	82	98	65	76	78	104	66	79
7	Vinayagam	29	M	55	76	120	84	96	82	129	74	92	76	119	69	86	74	112	68	83	75	115	73	87
8	Saroja	58	F	50	84	130	86	101	84	134	88	103	81	126	74	91	76	112	68	83	78	114	69	84
9	Sarisa	34	F	55	86	119	75	90	86	125	79	94	82	111	68	82	76	103	68	80	77	110	69	83
10	Kasiammal	49	F	60	85	135	84	101	86	134	84	101	74	119	71	87	72	122	94	103	75	125	75	92
11	Jaya	20	F	56	84	121	82	95	86	124	85	98	84	119	72	88	87	117	73	88	85	121	74	90
12	Kanniyammal	55	F	54	84	125	86	99	86	126	87	100	85	122	84	97	79	110	78	89	82	114	82	93
13	Nagalakshmi	25	F	50	86	122	79	93	88	126	84	98	82	122	79	93	69	103	69	80	72	105	72	83
14	Suganya	20	F	40	88	110	70	83	90	115	69	84	86	120	84	96	74	104	69	81	69	104	71	82
15	Noorjahan	42	F	70	84	132	85	101	86	135	84	101	90	143	92	109	81	127	84	98	71	112	78	89
16	Vennila	29	F	55	82	119	74	89	86	124	85	98	76	96	69	78	74	108	74	85	76	112	76	88
17	Nandakumar	27	M	62	86	122	84	97	88	126	86	99	87	120	85	97	74	118	79	92	76	109	69	82
18	Usha	28	F	50	85	119	76	90	82	126	87	100	84	122	79	93	79	106	68	81	71	105	71	82
19	Dhanalaxmi	40	F	55	88	126	88	101	92	130	84	99	84	121	79	93	80	119	72	88	81	122	81	95
20	Muthammal	58	F	55	84	134	86	102	88	136	89	105	81	122	76	91	84	121	72	88	85	124	74	91

DATA OF PATIENTS - INTRAOP – BUPIVACAINE GROUP

S No	20				25				30				45				60				75			
	HR	SBP	DBP	MAP	HR	SBP	DBP	MAP	HR	SBP	DBP	MAP	HR	SBP	DBP	MAP	HR	SBP	DBP	MAP	HR	SBP	DBP	MAP
1	82	114	70	85	84	109	69	82	85	122	74	90	86	126	78	94	82	120	74	89	88	117	68	84
2	83	128	77	94	85	125	74	91	88	132	82	99	86	128	79	95	87	122	74	90	84	124	75	91
3	79	122	75	91	78	124	73	90	82	126	78	94	75	119	72	88	80	124	74	91	91	130	79	96
4	82	102	60	74	84	101	71	81	85	111	76	88	87	115	79	91	79	112	77	89	84	107	77	87
5	81	110	74	86	84	114	75	88	86	113	72	86	83	112	71	85	82	108	68	81	79	113	72	86
6	82	111	69	83	84	114	73	87	86	115	72	86	88	119	76	90	84	117	75	89	85	122	79	93
7	77	117	75	89	79	107	77	87	82	114	79	91	85	116	70	85	79	111	73	86	76	114	75	88
8	74	118	73	88	84	122	74	90	79	117	72	87	82	122	76	91	77	124	78	93	76	126	84	98
9	75	112	72	85	78	114	75	88	82	119	81	94	85	116	82	93	77	122	86	98	79	126	86	99
10	76	121	73	89	74	120	72	88	72	119	73	88	77	116	72	87	82	125	76	92	78	129	82	98
11	86	123	75	91	84	125	74	91	86	122	75	91	87	123	84	97	88	124	75	91	86	122	74	90
12	76	116	78	91	77	118	79	92	75	122	82	95	74	123	83	96	82	124	84	97	79	125	85	98
13	74	110	76	87	77	112	82	92	82	114	84	94	79	116	82	93	77	122	86	98	82	134	92	106
14	76	111	72	85	74	114	73	87	72	108	71	83	74	109	72	84	68	114	81	92	70	121	88	99
15	74	117	79	92	73	103	69	80	74	93	52	66	84	97	62	74	72	117	74	88	81	109	61	77
16	78	114	78	90	82	115	82	93	85	116	78	91	84	118	82	94	87	122	84	97	85	126	86	99
17	77	114	72	86	74	113	68	83	76	114	72	86	82	116	76	89	74	118	74	89	77	122	76	91
18	76	111	72	85	75	114	73	87	77	121	77	92	74	119	75	90	72	122	81	95	73	125	84	98
19	82	124	83	97	83	122	84	97	85	125	86	99	87	121	84	96	86	126	89	101	85	122	84	97
20	87	126	81	96	86	126	85	99	79	127	89	102	84	129	91	104	82	124	89	101	86	126	88	101

DATA OF PATIENTS - INTRAOP – BUPIVACAINE GROUP

S No	90				105				120				135				150				165			
	HR	SBP	DBP	MAP	HR	SBP	DBP	MAP	HR	SBP	DBP	MAP	HR	SBP	DBP	MAP	HR	SBP	DBP	MAP	HR	SBP	DBP	MAP
1	92	115	70	85	86	118	76	90	84	122	74	90	85	124	76	92	88	126	78	94				
2	82	127	84	98																				
3	84	122	74	90	82	121	71	88	86	126	78	94												
4	85	106	76	86	86	116	70	85	88	115	87	96												
5	84	120	82	95	84	117	83	94	86	130	86	101												
6	82	124	82	96	86	121	79	93	86	126	85	99												
7	74	116	74	88	79	121	78	92	82	122	81	95	76	119	79	92	84	124	85	98				
8	74	130	82	98	84	128	83	98	85	129	84	99	82	127	84	98	86	132	86	101				
9	88	128	85	99																				
10	79	130	84	99	76	128	83	98	86	126	85	99	79	127	84	98	79	128	85	99	86	129	86	100
11	87	123	75	91	85	127	77	94	86	129	84	99	85	127	86	100								
12	77	121	86	98	82	122	84	97	84	126	85	99												
13	85	135	88	104	86	134	86	102	85	129	84	99	87	128	82	97								
14	69	130	84	99	74	110	72	85	80	117	75	89												
15	88	132	85	101	85	137	92	107	78	117	88	98	77	110	88	95	70	116	79	91	71	118	75	89
16	84	130	92	105	82	128	87	101	86	128	86	100												
17	78	124	82	96	80	125	85	98	84	126	86	99												
18	75	126	85	99	86	124	81	95	86	125	84	98												
19	85	126	86	99																				
20	87	125	88	100	88	127	89	102	89	131	92	105	87	133	91	105	86	129	90	103				

DATA OF PATIENTS - INTRAOP – BUPIVACAINE + CLONIDINE GROUP

S No	Name	Age	Sex	Wt	BHR	BSBP	BDBP	BMAP	0				5				10				15			
									HR	SBP	DBP	MAP	HR	SBP	DBP	MAP	HR	SBP	DBP	MAP	HR	SBP	DBP	MAP
1	Sasikala	28	F	50	86	120	82	95	88	126	88	101	87	122	84	97	82	112	71	85	78	110	69	83
2	Rani	42	F	55	82	130	85	100	84	136	90	105	80	128	74	92	79	119	69	86	82	124	72	89
3	Dhanabakyam	45	F	55	88	128	85	99	90	136	84	101	82	129	82	98	79	119	71	87	74	104	68	80
4	Anjalachi	40	F	50	84	127	84	98	90	130	86	101	84	122	79	93	86	124	74	91	85	122	69	87
5	Saroja	59	F	52	88	134	85	101	90	140	92	108	88	130	84	99	86	129	85	100	78	128	84	99
6	Jayanthi	20	F	54	78	118	72	87	82	122	84	97	69	109	71	84	68	98	69	79	72	97	70	79
7	Nirmala	27	F	54	88	127	84	98	86	130	86	101	84	121	79	93	79	118	69	85	69	122	72	89
8	Maheswari	45	F	60	80	125	84	98	88	131	92	105	74	120	72	88	76	104	69	81	78	102	68	79
9	Ammu	20	F	50	86	117	74	88	90	120	81	86	119	110	72	85	84	98	69	79	79	94	68	77
10	Manimegala	41	F	55	84	132	84	100	88	140	91	107	84	130	84	99	80	128	79	95	79	121	72	88
11	Kuppammal	31	F	55	82	126	85	99	88	130	88	102	84	120	72	88	79	119	69	86	72	104	72	83
12	Jasintha	40	F	50	86	129	88	102	110	121	88	99	100	130	91	104	94	126	85	99	83	119	84	96
13	Muniyammal	46	F	53	88	120	80	93	86	110	80	90	80	112	85	94	79	104	76	85	73	95	66	76
14	Parimala	24	F	55	84	125	85	98	90	140	90	107	78	117	73	88	74	101	68	79	77	117	71	86
15	Laila	42	F	64	86	128	84	99	86	130	85	100	70	122	92	102	72	122	87	99	71	97	60	72
16	Adhilaxmi	35	F	50	84	114	76	89	101	116	74	88	95	95	54	68	73	105	70	82	81	115	70	85
17	Indrani	40	F	40	82	135	86	102	70	132	92	105	72	110	97	101	82	93	55	68	76	107	72	84
18	Vanitha	24	F	55	110	110	84	93	120	111	73	86	95	87	50	62	83	108	70	83	90	102	58	73
19	Malliga	55	F	54	86	120	80	93	80	120	79	93	70	90	52	65	72	91	60	70	68	94	65	75
20	Loganayaki	20	F	50	86	126	84	98	88	130	85	100	84	120	84	96	82	119	79	92	79	118	78	91

DATA OF PATIENTS - INTRAOP – BUPIVACAINE + CLONIDINE GROUP

S No	20				25				30				45				60				75			
	HR	SBP	DBP	MAP	HR	SBP	DBP	MAP	HR	SBP	DBP	MAP	HR	SBP	DBP	MAP	HR	SBP	DBP	MAP	HR	SBP	DBP	MAP
1	76	114	74	87	75	102	71	81	74	114	74	87	76	112	76	88	78	118	75	89	80	120	74	89
2	76	124	75	91	71	126	74	91	74	129	76	94	80	130	82	98	82	129	81	97	86	128	82	97
3	75	110	72	85	72	112	74	87	76	115	78	90	80	120	81	94	84	122	82	95	86	118	79	92
4	84	118	69	85	82	117	68	84	79	120	71	87	84	122	76	91	82	124	75	91	86	129	78	95
5	74	129	86	100	84	130	82	98	82	132	84	100	82	129	85	100	84	133	86	102	82	128	85	99
6	71	108	71	83	74	110	72	85	76	106	71	83	78	108	72	84	82	110	74	86	84	118	76	90
7	74	112	69	83	72	114	68	83	76	112	71	85	84	126	76	93	82	124	75	91	86	128	78	95
8	82	110	74	86	86	112	75	87	78	114	76	89	82	124	82	96	79	126	81	96	83	124	81	95
9	78	103	69	80	76	108	71	83	74	108	72	84	72	110	74	86	74	112	75	87	71	119	82	94
10	84	124	74	91	76	122	73	89	78	121	75	90	84	119	69	86	82	124	73	90	88	124	76	92
11	69	114	74	87	74	118	75	89	75	122	78	93	84	126	84	98	86	122	81	95	82	121	82	95
12	80	116	75	89	78	114	81	92	74	120	80	93	78	121	74	90	76	121	80	94	70	106	72	83
13	66	100	70	80	66	110	77	88	67	105	72	83	66	98	69	79	64	94	64	74	68	107	73	84
14	68	101	65	77	74	107	65	79	76	113	77	89	88	105	66	79	90	133	94	107	88	112	69	83
15	74	109	69	82	75	14	72	53	73	113	73	86	72	110	72	85	76	114	75	88	81	118	74	89
16	85	112	64	80	88	118	84	95	82	121	78	92	84	113	71	85	86	120	71	87	88	112	76	88
17	82	110	73	85	78	124	92	103	77	125	95	105	76	123	86	98	82	113	85	94	85	127	90	102
18	75	101	71	81	77	102	66	78	76	102	69	80	77	111	76	88	76	115	79	91	76	112	77	89
19	66	102	70	81	68	103	72	82	66	117	79	92	64	113	75	88	71	120	85	97	74	43	79	67
20	78	121	76	91	72	117	74	88	69	108	72	84	74	110	69	83	75	114	76	89	76	116	78	91

DATA OF PATIENTS - INTRAOP – BUPIVACAINE + CLONIDINE GROUP

S No	90				105				120				135				150				165			
	HR	SBP	DBP	MAP	HR	SBP	DBP	MAP	HR	SBP	DBP	MAP	HR	SBP	DBP	MAP	HR	SBP	DBP	MAP	HR	SBP	DBP	MAP
1	88	122	76	91																				
2	88	127	81	96	88	126	79	95	90	128	86	100	88	126	85	99	90	128	86	100				
3	78	120	81	94	88	124	82	96	88	128	86	100												
4	85	130	82	98	84	126	79	95	82	124	74	91	88	122	75	91	90	128	81	97				
5	85	130	84	99																				
6	78	112	74	87	86	114	78	90	88	124	84	97												
7	79	130	81	97	84	124	79	94	86	130	82	98												
8	72	121	76	91	74	122	78	93	80	128	86	100												
9	69	122	83	96	74	118	82	94	78	122	84	97	88	126	88	101								
10	92	129	78	95	86	132	84	100	84	128	82	97	82	133	88	103	88	126	79	95	86	131	84	100
11	83	122	84	97	84	126	85	99	90	126	85	99												
12	74	118	75	89	76	126	69	88																
13	73	112	74	87																				
14	97	119	78	92	100	124	85	98	82	127	84	98	78	121	85	97	78	130	84	99				
15	84	122	76	91																				
16	87	118	68	85	73	114	69	84	75	121	73	89	91	123	79	94	90	121	80	94				
17	76	132	95	107	74	131	93	106	82	139	86	104	84	129	71	90	85	120	66	84				
18	82	117	80	92	87	114	71	85	68	111	71	84	77	119	80	93	70	115	87	96				
19	69	117	76	90	72	115	78	90	74	122	87	99	78	119	73	88								
20	82	115	79	91	79	122	84	97	84	122	86	98												

DATA OF PATIENTS - POSTOP – BUPIVACAINE GROUP

S No	0 HOUR						0.5 HOUR						1 HOURS						1.5 HOURS					
	HR	SBP	DBP	MAP	VAS	SS	HR	SBP	DBP	MAP	VAS	SS	HR	SBP	DBP	MAP	VAS	SS	HR	SBP	DBP	MAP	VAS	SS
1	88	126	78	94	0	0	86	120	76	91	1	0	84	122	75	91	2	0	85	126	74	91	3	0
2	86	130	82	98	0	1	84	129	79	96	0	0	79	133	84	100	1	0	78	126	82	97	1	0
3	90	130	84	99	0	0	82	126	82	97	0	0	80	124	81	95	0	0	82	130	84	99	1	0
4	75	120	84	96	0	0	77	113	72	86	0	0	76	114	76	89	0	0	82	116	75	89	1	0
5	88	128	84	99	0	0	84	122	81	95	0	0	82	120	79	93	1	0	85	122	81	95	1	0
6	84	122	82	95	0	0	82	117	78	91	0	0	86	121	81	94	0	0	84	122	84	97	0	0
7	76	120	82	95	0	0	74	119	79	92	0	0	72	119	81	94	0	0	75	122	81	95	1	0
8	86	120	84	96	0	0	84	122	85	97	0	0	78	119	84	96	0	0	76	126	86	99	1	0
9	86	120	84	96	0	0	85	117	79	92	0	0	79	116	78	91	0	0	78	112	74	87	1	0
10	84	126	85	99	0	0	82	122	87	99	0	0	85	123	82	96	1	0	86	124	82	96	1	0
11	86	125	84	98	0	0	84	122	85	97	0	0	82	126	84	98	1	0	84	127	88	101	1	0
12	82	122	84	97	0	0	80	126	85	99	0	0	78	127	89	102	1	0	79	126	84	98	1	0
13	86	122	85	97	0	0	85	117	79	92	0	0	84	116	78	91	1	0	79	119	82	94	1	0
14	80	115	75	88	0	1	82	118	69	85	0	0	84	120	71	87	1	0	86	120	74	89	1	0
15	92	130	80	97	0	0	88	122	76	91	0	0	86	124	76	92	1	0	84	124	74	91	1	0
16	86	121	85	97	0	0	84	119	84	96	0	0	82	116	77	90	1	0	79	118	78	91	1	0
17	80	120	82	95	0	0	82	119	79	92	0	0	84	117	75	89	0	0	79	119	77	91	1	0
18	87	124	82	96	0	0	86	119	69	86	0	0	85	119	72	88	1	0	82	122	75	91	1	0
19	84	122	84	97	0	0	82	119	79	92	0	0	80	126	85	99	1	0	80	127	84	98	1	0
20	84	128	84	99	0	0	86	131	85	100	0	0	88	132	86	101	1	0	85	133	84	100	1	0

DATA OF PATIENTS - POSTOP – BUPIVACAINE GROUP

S No	2 HOURS						2.5 HOURS						3 HOURS						3.5 HOURS					
	HR	SBP	DBP	MAP	VAS	SS	HR	SBP	DBP	MAP	VAS	SS	HR	SBP	DBP	MAP	VAS	SS	HR	SBP	DBP	MAP	VAS	SS
1																								
2	82	128	85	99	2	0	84	127	84	98	2	0	85	128	86	100	2	0	86	126	85	99	3	0
3	78	132	82	99	1	0	80	128	84	99	2	0	82	128	86	100	2	0	84	126	84	98	2	0
4	87	115	74	88	2	0	77	118	76	90	2	0	82	120	76	91	2	0	85	119	74	89	3	0
5	79	124	83	97	2	0	78	121	84	96	2	0	80	123	83	96	3	0						
6	85	123	86	98	1	0	84	125	84	98	1	0	85	121	81	94	1	0	84	122	83	96	2	0
7	73	123	82	96	1	0	72	122	83	96	1	0	74	121	82	95	2	0	75	124	81	95	2	0
8	75	130	88	102	1	0	77	32	86	68	1	0	74	129	85	100	2	0	77	127	84	98	2	0
9	82	114	72	86	1	0	80	110	73	85	1	0	81	111	74	86	2	0	82	112	75	87	2	0
10	87	119	80	93	1	0	86	126	82	97	2	0	85	130	84	99	2	0	84	129	83	98	3	0
11	85	122	81	95	1	0	84	119	76	90	2	0	82	123	79	94	2	0	83	124	81	95	2	0
12	77	124	85	98	1	0	81	122	86	98	2	0	82	127	85	99	2	0	82	126	84	98	2	0
13	81	117	81	93	2	0	82	116	71	86	2	0	83	115	72	86	2	0	84	116	74	88	2	0
14	84	120	75	90	1	0	85	122	76	91	2	0	82	123	74	90	2	0	83	124	73	90	2	0
15	82	120	72	88	2	0	84	122	76	91	2	0	86	126	78	94	3	0						
16	77	120	82	95	2	0	76	122	82	95	2	0	78	123	84	97	2	0	82	125	85	98	3	0
17	77	121	78	92	1	0	78	123	81	95	1	0	76	122	80	94	2	0	75	124	79	94	2	0
18	77	123	74	90	1	0	79	121	71	88	1	0	82	122	74	90	2	0	75	123	75	91	2	0
19	81	125	84	98	2	0	82	126	83	97	2	0	79	127	84	98	2	0	82	122	82	95	2	0
20	87	131	85	100	2	0	85	132	87	102	2	0	86	133	86	102	2	0	87	131	87	102	3	0

DATA OF PATIENTS - POSTOP – BUPIVACAINE GROUP

S No	4 HOURS						4.5 HOURS						5 HOURS						5.5 HOURS					
	HR	SBP	DBP	MAP	VAS	SS	HR	SBP	DBP	MAP	VAS	SS	HR	SBP	DBP	MAP	VAS	SS	HR	SBP	DBP	MAP	VAS	SS
1																								
2																								
3	84	122	84	97	3	0																		
4																								
5																								
6	82	122	84	97	2	0	83	125	84	98	2	0	85	126	85	99	3	0						
7	76	124	83	97	3	0																		
8	76	128	86	100	2	0	77	127	87	100	3	0												
9	83	116	76	89	2	0	84	115	75	88	2	0	84	118	74	89	3	0						
10																								
11	85	126	82	97	3	0																		
12	83	125	82	96	3	0																		
13	85	118	75	89	2	0	86	119	77	91	3	0												
14	87	120	71	87	2	0	86	122	72	89	2	0	85	124	73	90	2	0	84	125	74	91	2	0
15																								
16																								
17	74	126	84	98	3	0																		
18	77	122	76	91	2	0	82	124	75	91	3	0												
19	82	126	84	98	3	0																		
20																								

DATA OF PATIENTS - POSTOP – BUPIVACAINE GROUP

S No	6 HOURS						7 HOURS						Duration of analgesia (Minutes)
	HR	SBP	DBP	MAP	VAS	SS	HR	SBP	DBP	MAP	VAS	SS	
1													240
2													300
3													360
4													330
5													300
6													420
7													390
8													420
9													390
10													375
11													375
12													360
13													405
14	86	124	72	89	2	0	86	126	74	91	3	0	540
15													345
16													330
17													360
18													390
19													330
20													360

DATA OF PATIENTS - POSTOP – BUPIVACAINE + CLONIDINE GROUP

S No	0 HOUR						0.5 HOUR						1 HOURS						1.5 HOURS					
	HR	SBP	DBP	MAP	VAS	SS	HR	SBP	DBP	MAP	VAS	SS	HR	SBP	DBP	MAP	VAS	SS	HR	SBP	DBP	MAP	VAS	SS
1	86	126	76	93	0	1	80	124	74	91	0	1	84	120	72	88	0	1	82	112	72	85	1	1
2	90	130	86	101	0	1	88	124	84	97	0	1	82	122	80	94	1	1	84	122	82	95	1	1
3	90	130	84	99	0	1	86	126	80	95	0	1	88	124	80	95	0	1	84	120	82	95	0	0
4	86	130	80	97	0	1	88	126	76	93	0	1	84	122	74	90	0	1	82	120	75	90	0	0
5	88	133	85	101	0	1	86	130	84	99	0	1	84	132	85	101	0	0	85	133	84	100	0	0
6	86	120	85	97	0	1	84	117	79	92	0	1	79	118	81	93	0	0	78	117	79	92	1	0
7	86	128	84	99	0	1	84	122	79	93	0	1	82	120	74	89	0	0	79	120	72	88	1	0
8	88	124	82	96	0	1	86	122	79	93	0	1	82	121	78	92	0	0	78	120	74	89	0	0
9	80	124	82	96	0	1	76	120	81	94	0	1	74	119	79	92	0	0	72	118	82	94	0	0
10	78	126	84	98	0	1	76	122	79	93	0	1	82	119	72	88	1	0	84	118	74	89	1	0
11	88	128	86	100	0	1	84	126	85	99	0	1	86	122	84	97	0	0	74	123	81	95	0	0
12	80	124	82	96	0	1	84	120	82	95	0	0	82	124	84	97	0	0	83	126	86	99	1	0
13	74	110	70	83	0	1	80	112	74	87	0	1	76	114	74	87	1	0	79	113	75	88	1	0
14	79	120	67	85	0	1	76	122	74	90	0	0	79	120	72	88	0	0	81	124	73	90	0	0
15	86	126	82	97	0	1	84	119	78	92	0	1	82	122	81	95	0	0	76	123	83	96	1	0
16	90	124	82	96	0	1	86	123	84	97	0	1	82	125	86	99	0	0	79	124	83	97	0	0
17	84	111	75	87	0	1	82	107	64	78	0	0	76	105	76	86	1	0	78	107	74	85	1	0
18	80	113	72	86	0	1	84	103	61	75	0	1	86	107	77	87	0	0	82	106	76	86	0	0
19	80	124	88	100	0	1	76	120	86	97	0	1	78	120	84	96	1	1	80	119	93	102	1	0
20	88	122	84	97	0	1	84	125	80	95	0	1	78	130	84	99	0	1	76	129	85	100	1	0

DATA OF PATIENTS - POSTOP – BUPIVACAINE + CLONIDINE GROUP

S No	2 HOURS						2.5 HOURS						3 HOURS						3.5 HOURS					
	HR	SBP	DBP	MAP	VAS	SS	HR	SBP	DBP	MAP	VAS	SS	HR	SBP	DBP	MAP	VAS	SS	HR	SBP	DBP	MAP	VAS	SS
1	84	120	74	89	1	0	82	122	72	89	1	0	80	124	74	91	1	0	79	125	72	90	1	0
2	80	124	84	97	1	0	78	120	81	94	1	0	80	122	81	95	1	0	82	124	82	96	2	0
3	82	119	79	92	0	0	80	118	81	93	1	0	84	118	82	94	1	0	82	116	81	93	1	0
4	79	120	74	89	1	0	81	118	72	87	1	0	82	124	74	91	1	0	78	126	75	92	1	0
5	82	134	86	102	1	0	80	135	86	102	1	0	82	138	89	105	1	0	84	135	88	104	1	0
6	72	116	78	91	1	0	74	115	74	88	1	0	76	114	72	86	1	0	78	110	70	83	1	0
7	78	118	70	86	1	0	78	118	72	87	1	0	80	120	74	89	1	0	82	117	71	86	1	0
8	76	118	74	89	1	0	76	117	72	87	1	0	75	118	73	88	1	0	74	121	72	88	1	0
9	73	117	81	93	0	0	75	122	83	96	0	0	74	120	84	96	1	0	78	125	84	98	1	0
10	81	117	72	87	1	0	79	116	76	89	1	0	81	114	72	86	1	0	82	121	73	89	1	0
11	76	122	84	97	1	0	78	126	86	99	1	0	75	124	82	96	1	0	74	122	81	95	1	0
12	86	122	85	97	1	0	85	126	84	98	1	0	84	119	82	94	2	0	83	117	79	92	2	0
13	82	112	73	86	1	0	84	110	71	84	1	0	82	112	73	86	1	0	84	118	77	91	1	0
14	82	126	77	93	1	0	76	124	75	91	1	0	74	122	74	90	1	0	75	126	76	93	1	0
15	78	116	75	89	1	0	82	119	76	90	1	0	78	122	81	95	1	0	77	123	83	96	2	0
16	78	121	84	96	1	0	84	119	81	94	1	0	86	123	84	97	1	0	88	125	86	99	1	0
17	80	111	69	83	1	0	76	116	74	88	1	0	74	117	81	93	1	0	78	121	73	89	2	0
18	86	114	79	91	0	0	84	116	70	85	1	0	80	120	72	88	1	0	84	118	74	89	1	0
19	82	122	82	95	1	0	78	120	80	93	1	0	76	120	82	95	1	0	76	122	84	97	1	0
20	74	128	84	99	1	0	72	127	82	97	1	0	76	126	80	95	1	0	78	122	81	95	1	0

DATA OF PATIENTS - POSTOP – BUPIVACAINE + CLONIDINE GROUP

S No	4 HOURS						4.5 HOURS						5 HOURS						5.5 HOURS					
	HR	SBP	DBP	MAP	VAS	SS	HR	SBP	DBP	MAP	VAS	SS	HR	SBP	DBP	MAP	VAS	SS	HR	SBP	DBP	MAP	VAS	SS
1	78	119	69	86	1	0	78	120	71	87	1	0	78	117	71	86	1	0	80	116	69	85	2	0
2	82	118	79	92	2	0	84	116	74	88	2	0	82	120	75	90	2	0	80	122	76	91	2	0
3	85	118	78	91	1	0	83	120	82	95	2	0	84	122	84	97	2	0	86	122	85	97	2	0
4	82	124	74	91	1	0	84	122	72	89	1	0	82	129	84	99	1	0	82	130	85	100	2	0
5	85	132	84	100	1	0	83	129	79	96	1	0	83	128	81	97	1	0	81	130	83	99	1	0
6	76	112	71	85	1	0	78	114	72	86	1	0	72	112	69	83	1	0	74	114	74	87	2	0
7	82	114	70	85	1	0	84	112	73	86	1	0	82	112	74	87	2	0	79	111	71	84	2	0
8	72	124	74	91	2	0	74	123	73	90	2	0	75	119	71	87	2	0	76	118	69	85	2	0
9	80	122	82	95	1	0	76	117	79	92	1	0	72	118	80	93	1	0	74	122	79	93	2	0
10	86	122	74	90	1	0	85	121	75	90	1	0	84	119	72	88	2	0	82	112	69	83	2	0
11	72	120	82	95	1	0	73	119	79	92	1	0	76	118	78	91	2	0	75	117	81	93	2	0
12	86	116	81	93	2	0	88	115	79	91	2	0	87	116	78	91	2	0	88	114	74	87	2	0
13	80	115	76	89	2	0	80	114	75	88	2	0	82	112	73	86	2	0	84	113	72	86	2	0
14	72	123	75	91	1	0	76	124	77	93	2	0	77	124	73	90	2	0	79	121	76	91	2	0
15	75	119	79	92	2	0	84	117	78	91	2	0	83	118	79	92	2	0	82	116	75	89	2	0
16	84	124	87	99	2	0	82	123	86	98	2	0	83	122	84	97	2	0	85	123	85	98	2	0
17	76	110	76	87	2	0	72	108	70	83	2	0	74	108	75	86	2	0	76	107	79	88	2	0
18	82	117	73	88	1	0	84	119	75	90	1	0	84	122	74	90	1	0	85	120	76	91	1	0
19	78	124	85	98	1	0	80	120	84	96	1	0	82	117	82	94	1	0	84	119	81	94	2	0
20	82	120	79	93	1	0	84	118	76	90	1	0	78	117	75	89	1	0	76	119	72	88	2	0

DATA OF PATIENTS - POSTOP – BUPIVACAINE + CLONIDINE GROUP

S No	6 HOURS						7 HOURS						8 HOURS						9 HOURS					
	HR	SBP	DBP	MAP	VAS	SS	HR	SBP	DBP	MAP	VAS	SS	HR	SBP	DBP	MAP	VAS	SS	HR	SBP	DBP	MAP	VAS	SS
1	82	115	71	86	2	0	80	118	73	88	2	0	78	117	72	87	2	0	76	118	73	88	2	0
2	78	124	74	91	2	0	78	122	73	89	2	0	80	125	75	92	2	0	82	126	80	95	3	0
3	88	124	86	99	2	0	84	122	84	97	2	0	86	124	82	96	2	0	85	126	83	97	2	0
4	84	129	86	100	2	0	86	128	84	99	2	0	85	129	84	99	2	0	84	128	85	99	2	0
5	80	130	82	98	2	0	82	133	84	100	2	0	83	132	82	99	2	0	84	133	83	100	2	0
6	76	112	72	85	2	0	75	111	71	84	2	0	72	110	73	85	2	0	76	114	74	87	3	0
7	78	113	72	86	2	0	76	114	74	87	2	0	76	115	76	89	2	0	78	118	75	89	2	0
8	78	117	68	84	2	0	74	119	69	86	2	0	72	120	71	87	2	0	71	119	72	88	2	0
9	72	121	74	90	2	0	75	122	75	91	2	0	74	128	78	95	2	0	72	122	74	90	2	0
10	79	110	72	85	2	0	78	114	73	87	2	0	83	115	72	86	2	0	84	116	79	91	3	0
11	82	116	79	91	2	0	80	119	78	92	2	0	78	121	76	91	2	0	82	122	75	91	2	0
12	88	118	72	87	2	0	87	119	74	89	3	0												
13	86	116	75	89	2	0	88	117	74	88	3	0												
14	76	122	78	93	2	0	78	124	77	93	2	0	82	126	82	97	3	0						
15	81	118	76	90	2	0	83	122	81	95	2	0	85	124	79	94	2	0	84	122	81	95	2	0
16	86	121	84	96	2	0	88	119	79	92	2	0	84	118	75	89	2	0	82	121	77	92	3	0
17	76	115	84	94	2	0	78	114	70	85	2	0	80	121	70	87	2	0	80	122	65	84	2	0
18	82	120	74	89	2	0	84	122	78	93	2	0	86	120	76	91	2	0	85	122	75	91	2	0
19	84	119	79	92	2	0	82	121	81	94	2	0	80	122	82	95	2	0	82	124	84	97	3	0
20	78	114	74	87	2	0	74	113	72	86	2	0	76	112	71	85	2	0	74	117	74	88	2	0

DATA OF PATIENTS - POSTOP – BUPIVACAINE + CLONIDINE GROUP

S No	10 HOURS						11 HOURS						12 HOURS						Duration of analgesia (Minutes)
	HR	SBP	DBP	MAP	VAS	SS	HR	SBP	DBP	MAP	VAS	SS	HR	SBP	DBP	MAP	VAS	SS	
1	78	120	74	89	2	0	80	120	75	90	3	0							750
2																			690
3	84	124	82	96	3	0													720
4	82	128	84	99	2	0	84	130	82	98	3	0							810
5	85	132	82	99	2	0	86	134	84	101	3	0							750
6																			660
7	76	119	74	89	2	0	78	120	75	90	3	0							780
8	70	124	74	91	3	0													720
9	73	124	73	90	2	0	76	121	78	92	3	0							795
10																			705
11	84	126	76	93	3	0													720
12																			525
13																			510
14																			630
15	86	123	82	96	2	0	87	124	79	94	2	0	85	124	84	97	3	0	810
16																			690
17	82	111	64	80	2	0	82	108	65	79	2	0	86	110	68	82	3	0	870
18	86	124	72	89	2	0	88	126	78	94	3	0							810
19																			675
20	75	118	75	89	2	0	76	122	74	90	2	0	76	125	75	92	3	0	840